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Annals of
Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER.

VOL. XVII, 1908.

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PUBLISHED QUARTERLY,

By Jones H. Parker,
Mermod-Jaccard Building,
St. Louis, Mo.
1908.

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TABLE OF CONTENTS.

I.—The Optic Nerve and the Accessory Cavities of the Nose, by Prof. Dr. A. Onodi, Budapest. Translated by H. Strass. 1

II.—Observations on Ten Cases of Intracranial Complications of Middle Ear Suppuration, by S. J. Kopetzky, M. D., New York. 117

III.—Ocular Symptoms of Intracranial Complications in Otitic Disease, by Percy Fridenberg, M. D., New York. .140

IV.—A Case of Thrombosis of the Posterior Cerebral Veins and Artery, Encephalitis, Purulent Leptomeningitis, Lateral Sinus Phlebitis and Parietal Thrombosis Following Mastoiditis, by W. Sohier Bryant, A. M., M. D., New York. 163

V.—A Plea for Conservatism in the Treatment of Certain Affections of the Nose and Ear, by Harry L. Myers, M. D., Norfolk, Va. 167

VI.—Primary Nasal Diphtheria, by Thomas Hubbard, M. D., Toledo, Ohio. 172

VII.—A Plea for Local Anesthesia in Operations on the Maxillary Sinus, by Gordon King, M. D., New Orleans. .178

VIII.—Contribution to the Study of Tuberculosis of the Nasal Mucosa—An Etiologic and Clinical Study, by Henri Caboche, M. D. Translated by Albert Miller, A. B., M. D. 180

IX.—Surgery of the Facial Nerve, by Joseph C. Beck, M. D., Chicago 265

X.—The Operation for Thrombosis of the Sigmoid Sinus and Internal Jugular Vein of Otitic Origin, by FRANK ALLPORT, M. D., Chicago....333

XI.—Treatment of the Infective Labyrinthitis After Fifteen Years' Experience, by DR. ALBERT JANSEN, Berlin...367

XII.—Pathology and Bacteriology of Suppurative Diseases of the Temporal Bone, by E. DE W. WALES, M. D., Indianapolis....398

XIII.—Operation for Brain Abscess of Otitic Origin, by J. A. STUCKY, M. D., Lexington, Ky....427

XIV.—Causation of the Suppuration of the Temporal Bone, by WILLIAM E. SAUER, M. D., St. Louis....431

XV.—A Report of Atypical and Typical Cases of Sinus Thrombosis Complicating Mastoiditis, by W. R. DABNEY, M. D., Marietta, Ohio....439

XVI.—Diagnosis and Treatment of Suppuration of the Labyrinth, by R. BISHOP CANFIELD, M. D., Ann Arbor, Michigan....451

XVII.—Chondroma and Osteoma in the Faucial Tonsils, by LORENZO NELSON GROSVENOR, M. D., Chicago....470

XVIII.—Developmental Absence of the Outer Right Sphenoidal Wall Occupied by a Vein Communicating Directly with the Cavernous Sinus. Operative Fatality and Autopsy, by FRANCIS P. EMERSON, M. D., Boston....479

XIX.—Report of a Case in Which Deficient Cerebration was a Prominent Symptom in Otitis Media Purulenta Chronica—Cured by a Radical Mastoid Operation, by D. W. LAYMAN, M. D., Indianapolis....487

XX.—The Treatment of Intumescent Rhinitis by a Submucous Method, by HENRY HORN, M. D....490

XXI.—Recurrent and Abductor Paralysis of the Larynx. Introductory Remarks on Anatomy and Physiology. Etiology of Paralyses of Central Origin, by J. W. Gleitsmann, M. D., New York 587

XXII.—Recurrent and Abductor Paralysis of the Larynx. Diagnosis and Treatment, by W. E. Casselberry, M. D., Chicago 607

XXIII.—The Etiology of Paralysis of the Recurrent Laryngeal Nerves of Peripheral Origin, by D. Bryson Delavan, M. D., New York....................................631

XXIV.—The Etiology of Otosclerosis, by Thomas J. Harris, M. D., New York....................................645

XXV.—Pathology and Prognosis of Chronic Progressive Hardness of Hearing—Otosclerosis, by W. Sohier Bryant, A. M., M. D., New York....................................652

XXVI.—Symptoms and Diagnosis of Otosclerosis, by Geo. E. Shambaugh, M. D., Chicago....................................661

XXVII.—The Non-Acoustic Functions of the Labyrinth: A Review of our Present Knowledge of the Organs of Static and Dynamic Equilibrium, by Percy Fridenberg, M. D., New York 670

XXVIII.—Chronic Recurrent Suppurative Otitis Media and its Relation to Mastoid and Intracranial Complications, by S. MacCuen Smith, M. D., Philadelphia, Pa...........723

XXIX.—Some Surgical Principles Essential to the Cure of Frontal Sinus Empyemas, by John F. Barnhill, M. D., Indianapolis 736

XXX.—A Case of Acute Suppuration of the Labyrinth Following Acute Otitis Media—Operation; Recovery, by Edward Bradford Dench, M. D., New York..................746

XXXI.—Black Hairy Tongue (Lingua Villosa Nigra), by N. RH. BLEGVAD, M. D., Copenhagen..................753

XXXII.—Papilloma of the Larynx in Children; a Further Consideration of the Subject, by J. PAYSON CLARK, M. D..787

XXXIII.—The Diagnosis of the Otitic Brain Abscess, by DR. THEODOR HEIMANN, Warsaw......................800

XXXIV.—The Importance of the Upper Respiratory Tract in the Etiology of Cryptogenetic Infections, Especially in Relation to Pleuritis, by GEO. B. WOOD, M. D., Philadelphia..855

XXXV.—The Morphology of the Turbinals, by J. M. INGERSOLL, A. M., M. D., Professor of Oto-Laryngology in the Medical Department of Western Reserve University, Cleveland 901

XXXVI.—Giant Tumors of the Larynx, by PROF. F. MASSEI, Naples 931

XXXVII.—The Operative Treatment of Malignant Tumors of the Nose, by PROF. DR. ALFRED DENKER, Erlangen....959

XXXVIII.—Cysts, Abscesses and Edema of the Epiglottis, by HENRY L. SWAIN, M. D., Professor of Otology and Laryngology at Yale, New Haven...........................974

XXXIX.—Some Interesting Cases of Diseases of the Alveolar Process, by W. H. HASKIN, M. D., New York........985

XL.—Submucous Resection in Ozena, by PROF. MERMOD, Lausanne 995

XLI.—The Treatment of Tuberculosis of the Upper Air Passages, by DR. THEODOR HERYNG, Warsaw. Translated by CLARENCE LOEB, M. D., St. Louis....................1000

XLII.—The Influence of Adrenalin in the Causation of Arterio-Sclerosis, by F. E. HOPKINS, M. D., Springfield, Mass. 1022

Abstracts from Current Otologic, Rhinologic and Laryngologic Literature:

I.—Ear 243, 495, 841, 1026
II.—Nose and Accessory Cavities 256, 499, 849, 1045
III.—Pharynx and Mouth 260, 505, 851, 1057
IV.—Larynx 259, 502, 850, 1064
V.—Miscellaneous 261, 852
Book Reviews 1072

ANNALS

OF

OTOLOGY, RHINOLOGY

AND

LARYNGOLOGY.

VOL. XVII. MARCH, 1908. No. 1.

I.

THE OPTIC NERVE AND THE ACCESSORY CAVITIES OF THE NOSE.

CONTRIBUTION TO THE STUDY OF CANALICULAR NEURITIS AND ATROPHY OF THE OPTIC NERVES OF NASAL ORIGIN.*

BY PROF. DR. A. ONODI, BUDAPEST.

TRANSLATED BY H. STRASS.

PREFACE.

For ten years I have been investigating the more delicate construction of the accessory cavities, and the relation of the optic nerve to them. The results obtained furnish the anatomic foundation for the theory of sight disturbances and blindness caused by diseases of the accessory cavities, particularly of the posterior ethmoid cells and the sphenoid sinus. An exact pathologico-anatomic foundation for this theory is still to be created. The observations with which we are familiar I have incorporated in the chapter on clinical observations.

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*This paper is translated with the authority of Professor Onodi given to the editor of the Annals of Otology, Rhinology and Laryngology.

The many discrepancies in our knowledge of the subject are mentioned, but the complete establishment of this theory is to be expected from exact clinical observations and pathologico-anatomic research. I offer my colleagues the work of many years in the hope that it will lead to further mutual research in rhinologic and ophthalmologic fields, and to the successful solution of this important question.

My preparations are photographed in natural size, ten of which I used for demonstration before the 33rd Congress of the Ophthalmological Society, at Heidelberg in 1906.

Of the 33 illustrations 8 preparations are used which appear in my atlas on "The Accessory Cavities of the Nose."

TOPOGRAPHIC ANATOMY.

The intimate relation of the optic nerve to the accessory cavities of the nose, and the more delicate topographico-anatomic relations have not, until recently, been exhaustively treated. Generally speaking, the text-books on anatomy pointed simply to the intimate relation of the optic nerve to the sphenoid sinus, which is mentioned, of course, in the description of the course of the optic nerve, and the base of the skull cavity. Berger and Tyrmann[1], in their monograph, speak of the relation of the optic nerve to the sphenoid sinus, and show three instances of the different thicknesses of the sphenoid sinus wall in the region of the foramen opticum. They also mention two skulls, in which the ethmoid labyrinth extends so far back, that the front wall of the sphenoid sinus lies behind the optic nerve canal, so that the latter does not touch the sphenoid sinus. Zuckerkandl[2] and Douglas[3] have described the sphenoid cell in the region of the lesser sphenoid wing. These were the forerunners of my years of introductory and preliminary research. In my publications I have shown the frequently intimate relation of the optic nerve to the last ethmoid cell, and proved in this way that it is about time to drop the almost mechanical assumption of the relation of the optic nerve to the sphenoid sinus. My completed studies have resulted in the establishment and confirmation of 38 different morphologic relations, bearing on the relation of the optic nerve to the sphenoid sinus, and the last ethmoid cell. When we take into consideration the embryonic relation of the canalis and sulcus opticus, and give our attention to the morphologic relations which the different parts of the sphenoid, such as the presphenoid, the orbitosphenoid, and the basisphenoid show in the fetal and post-fetal periods to the development of the sphenoid sinus, the appearance of the sphenoid turbinate, the development of the ethmoid cells, the frontal, and the appearance of centers of ossification (morphologic relations which may show deviations in the fetal and post-fetal period), then we can properly understand most of the morphologic relations which have been found and described, and explain their peculiarities. We do not wish in this connection to examine the morphologic relations of the optic nerves more closely; it is sufficient to throw light upon the topographic relations of the optic nerves, the canalis opticus, and sulcus opticus, and the close relation of this portion to the last ethmoid cell and to the sphenoid sinus.

The 38 different morphologic relations are divided into twelve groups: the individual ones are described with cuts of preparations made in natural size from photographs. The position and course of the optic nerve in the cranium are first presented and illustrated, then the region of the foramen opticum and the canalis opticus, and finally the different relations of the optic nerve, the sulcus opticus, the foramen opticum, and the canalis opticus to the last ethmoid cell, and the sphenoid sinus.

The region of the foramen opticum is illustrated from a bone preparation by figure 1. Above is seen the fissura orbitalis superior with the lesser wing of the sphenoid, ala parva, on the medial side of which is the foramen opticum, which is continuous with the short canalis opticus. The superior portion of the foramina optica is limited by an inferior border, the limbus sphenoidalis, which marks the boundary of the jugum sphenoidale lying anterior. Between the foramina optica, the limbus sphenoidalis, and the hypophyseal groove, the sella turcica limiting the tuberculum sellae, the sulcus opticus or sulcus chiasmatis may be seen.

Figure 2 shows the optic nerve entering the foramen opticum, the chiasm, and the optic tract. The flattened strands of this optic tract unite and cross in the chiasm between the limbus sphenoidalis and the tuberculum sellae in the sulcus opticus. The optic nerves emerge from the chiasm in order to pass above the ophthalmic artery in the foramen opticum. The figure shows the internal carotid going to the brain, and the origins of the oculomotorius, trochlearis, trigeminus and abducens.

Figures 2 and 104 in my Atlas[4] show the base of the brain with the attached tractus opticus in situ. Figure 2 shows in sagittal section the position and relation of the tractus opticus to sphenoid sinus. Fig 104 illustrates a preparation in sagittal section which was injected with a ten per cent. formol solution in order to maintain the mucosa of the individual cells in bladder-like form. The maxillary sinus, the frontal sinus, the ethmoid cells and the sphenoid sinus may be seen in situ, and the base of the brain with the attached tractus opticus and its relations to the ethmoid cells.

Figure 3 illustrates in a formol preparation all the structures which appertain to the orbital cavity. The bony wall has been removed, and the mucosa of the accessory nasal cavities may be seen in bladder-like shape, the inferior surface of the frontal lobe of the brain, the optic nerve and the internal carotid in situ. The position of the optic nerve and its close relation to the sphenoid may be observed under the frontal lobe.

Figure 4 illustrates, in a formol preparation, the position of the chiasm, the course of the left optic nerve, as far as the entrance into the bulb. The left optic nerve touches the left sphenoid sinus, otherwise the nerves have no relation with the accessory cavities in their course. The individual accessory cavities may be seen in bladder-like shape in situ, and on one side the mucosa of the roof of the nose, and, on the other, the opened nasal cavity and the relation of the nasal cavities to the periorbita.

Figure 5 illustrates the accessory cavities in situ, in a formol preparation, sagittal section, with the nasal cavity opened from without, and the relation of the optic nerve to the last ethmoid cell, and to the sphenoid sinus. Before we enter into a closer description of the individual morphologic relations it should be stated that in those cases in which the wall of the canalis opticus and of the sulcus opticus is formed by the last ethmoid cell, its wall may be extremely thin, as thin as tissue paper. In one case the partition was 1 mm. thick, another time 2 mm. In those cases in which the wall of the canalis opticus and of the sulcus opticus is formed by the wall of the sphenoid sinus, it was found with various thicknesses, ranging as follows: 1, 2, 3, 4, 5, 9 and 12 mm. We shall later discuss the importance of this fact, and likewise the bony dehiscence of the canalis opticus and the course of the canal in the sphenoid sinus and in the last ethmoid cell. The length of the canal may reach 6, 8, 10 or 12 mm.

GROUP I.

1. To this group belong those cases in which the posterior ethmoid cell and the sphenoid sinus are not in close relation with the canalis opticus or sulcus opticus. Figure 77 of the Atlas, already mentioned, shows a preparation in frontal section. The right sphenoid sinus is 12 mm. long, 10 mm. broad, and 9 mm. in height, and the left 9 by 10 by 20 mm. The ostium sphenoidale on each side is 1 mm. The bony stratum between the optic nerves, canalis opticus and sphenoid sinus has a thickness of 8 to 12 mm.

Figure 6 likewise shows a preparation in frontal section in which the bone substance, 6 mm. in thickness, separates the optic nerve from the sphenoid sinus and ethmoid cell. The optic nerve, the sphenoid sinus and maxillary sinus may be seen on the surface of the section.

Figure 76 in our Atlas shows a frontal section at the posterior border of the nasal septum, in which bone substance, 8 mm. in thickness, separates the sphenoid sinus from the hypophysis, above which both optic tracts with the chiasm may be seen.

GROUP II.

The relation of the last ethmoid cell to the optic nerves on both sides may exhibit three forms.

2. The last ethmoid cells constitute on both sides the medial and inferior wall of the canalis opticus. This relation which is shown in figure 7, in frontal section, is the most instructive, as it shows that the sphenoid sinuses have no relation to the optic nerves, and that they lie under the ethmoid cells where their openings may also be seen. In this preparation the posterior ethmoid cells extend to the foramen opticum, and constitute on both sides the medial and inferior walls of the canalis opticus. The ostium sphenoidale and the reduced anterior wall of the sphenoid sinus may be seen beneath the inferior wall of the last ethmoid cell, near the median line. The thin septum between the sphenoid sinus and the posterior ethmoid cell is 20 mm. wide on the right side, and 15 mm. on the left.

3. The right posterior ethmoid cell constitutes the medial wall of the right canalis opticus, the left posterior ethmoid

cell constitutes the medial and inferior wall of the left canalis opticus, and the wall of the left third of the sulcus opticus.

4. The ethmoid cells are represented by a cavity, this is merged on the left side with the frontal, and these together form a common cavity. The figures 97 and 98 in the Atlas illustrate this preparation in transverse section. It deals with a structural anomaly, the tissue everywhere shows normal conditions, and the openings of the so-called cavities on both sides are evenly and systematically arranged. On the left the frontal merges with a cavity taking the place of one of the ethmoid cells; the length of this common cavity measures 62 mm. On the right the frontal is separated from the cavity representing the ethmoid cell; the cavity is 45 mm. long, the frontal is 23 mm. long. The anterior wall of the left frontal is 7 mm., that of the right one 1.5 mm. thick.

Separated from the anterior wall by 22 mm. lies the foramen ethmoidale anterius, which takes a downward course along the lateral wall of the frontal, continuing in a semicanalis ethmoidalis, 16 mm. in length. The greatest width of the cavity taking the place of the ethmoid cell is 25 mm. On the left, the frontal is 45 mm., on the right 40 mm. wide; on the left it is 65 mm., on the right 40 mm. in height. The frontal opens on both sides with an orifice 9 mm. long and 4 mm. wide above the anterior terminus of the hiatus semilunaris. The processus uncinatus is well developed; the bulla ethmoidalis well arched. The sphenoid sinus opens on the posterior part of the hiatus semilunaris. The superior turbinate arises as a projection from the middle turbinate; between them the ethmoid cell, bilaterally symmetrical, opens into the superior meatus through small round orifices. A sphenoid sinus exists on the right side. On the right side the frontal shows a recess, the cavity taking the place of the ethmoid cells, two recesses. The lamina cribrosa is 12 mm. long, 2 mm. wide, and constitutes the center of the roof of the nasal cavity. The cavity representing the right ethmoid cell constitutes the inferior and medial wall of the right canalis opticus, and the wall of the right and middle third of the sulcus opticus; the cavity representing the left ethmoid cell, which merges with the frontal, constitutes the medial wall of the left canalis opticus, and the wall of the left third of the sulcus opticus.

GROUP III.

The relations of the posterior ethmoid cell on one side to the optic nerve show seven forms, five of which concern the same side, two the opposite side.

5. On the same side the last ethmoid cell constitutes the medial, inferior and lateral wall of the canalis opticus, also the wall of the right third of the sulcus opticus. In the Atlas, figure 17 shows a bone preparation in the sagittal section, illustrating this relation. On the right side, one sees that the otherwise large sphenoid sinus bears no relation to the foramen opticum, canalis opticus or sulcus opticus. The sphenoid sinus is 22 mm. long, 18 mm. high, the posterior ethmoid cell is 30 mm. long; beginning at the median line, it is 18 mm. wide. The latter extends under the sulcus and the foramen opticum into the small sphenoid lobe, and constitutes the whole wall of the sulcus opticus, also the medial, inferior and lateral wall of the canalis opticus.

6. On the same side the posterior ethmoid cell constitutes the medial and inferior wall of the left canalis opticus, and the whole wall of the sulcus opticus.

In the Atlas, figure 16 shows a bone preparation in the sagittal section, illustrating this relation. On the left side one sees that the otherwise large sphenoid sinus stands in no relation to the foramen opticus, canalis opticus, or sulcus opticus. The sphenoid sinus is 25 mm. long, 15 mm. high, and 15 mm. wide, from the median line; the last ethmoid cell is 11 mm long, 14 mm. wide from the median line, and 13 mm. in height. The posterior ethmoid cell constitutes the entire wall of the sulcus opticus, also the medial and inferior wall of the canalis opticus.

7. On the same side the last ethmoid cell constitutes the inferior and medial wall of the canalis opticus. Figure 13 of the Atlas shows a bone preparation with the sagittal section illustrating this relation. On the left side is seen the last ethmoid cell, 34 mm. long and 16 mm. high, and its relation to the optic nerve; the ethmoid cell extends back 6 mm., until close under the canalis opticus. The accessory and nose cavities are opened from the outside.

8. The last ethmoid cell constitutes on the same side the upper, medial and inferior wall of the canalis opticus. Figures 8 and 9 illustrate this relation. Figure 8 shows by a sagittal section how the last right ethmoid cell surrounds the canalis opticus

medially, above and below. Figure 9 illustrates a preparation, a frontal section opening the last ethmoid cell in front; it was also opened through the nasal cavity. On the right side one sees the entrance of the optic nerve into the foramen opticum, next to the carotis interna, and the canalis opticus, 12 mm. long, taking its course into the last ethmoid cell, which surrounds it above, below and medially. On the same side the last ethmoid cell constitutes the superior, inferior, medial and lateral wall of the canalis opticus.

9. The last ethmoid cell forms on the same side the upper, lower, medial and lateral walls of the canalis opticus. Figure 10 illustrates this relation in a sagittal section, on the right side is seen the canalis opticus in the last ethmoid cell, the entrance and departure of the optic nerve into and out of the canalis opticus. The length of the canalis opticus is 10 mm.; it disappears into a posterior ethmoid cell 15 mm. long and 18 mm. in height.

10. The last ethmoid cell constitutes, on the same side, the medial wall of the canalis opticus. Figure 11 illustrates this relation in a sagittal section. On the left side is seen the entrance of the optic nerve into the canalis opticus; the thin partition wall, which surrounds the optic nerve medially, has been removed in the ethmoid cell; the optic nerve, for 6 mm. in length, touches the ethmoid cell.

11. The posterior ethmoid cell constitutes on one side the inferior wall of the canalis opticus. Figures 12 and 13 illustrate this relation. Figure 12 shows by a sagittal section on the left side the course of the optic nerve above the last ethmoid cell; its length is 7 mm.; the thin partition wall has been removed. Figure 13 illustrates by a frontal section the course of the optic nerve above the posterior ethmoid cell. Its length is 4 mm. The thin partition wall has been removed.

12. The right posterior ethmoid cell constitutes on both sides the medial and inferior wall of the canalis opticus, and the entire wall of the sulcus opticus. Figure 14 in the Atlas illustrates this relation, and figure 14 in corresponding sagittal sections. The right posterior ethmoid cell is 17 mm. high, 22 mm. long, and 38 mm. wide. The right sphenoid sinus 14 mm. high, 22 mm. long and 28 mm. wide. The right posterior ethmoid cell is of extraordinary dimensions, it lies above the sphenoid sinuses and bears an intimate relation to the chiasma and both nervi optici, because the entire sulcus opticus and the inferior wall

of the canalis opticus are separated from the posterior ethmoid cell by an exceedingly thin, bony wall. The left sphenoid sinus is 26 mm. long, 20 mm. wide and 14 mm. high.

13. The left posterior ethmoid cell constitutes the medial wall of the right canalis opticus. Figure 53 of the Atlas shows a preparation illustrating this relation in a sagittal section.

The sphenoid sinuses show an asymetry, owing to the fact that the left sphenoid sinus lies anterior to the right sphenoid sinus. The right sphenoid sinus is 18 mm. long, 15 mm. wide, 16 mm. in height; the left sphenoid sinus is 15 mm. long, 16 mm. wide, 17 mm. in height. The left posterior ethmoid cell is 16 mm. long, 15 mm. broad; it constitutes the medial wall of the right canalis opticus.

GROUP IV.

The sphenoid sinuses on both sides show a close relation to the optic nerves, illustrated by five morphologic relations.

14. The sphenoid sinuses constitute on both sides the medial and inferior wall of the canalis opticus, and the wall of the sulcus opticus. Figure 15 illustrates this relation in a formol preparation. The bony walls of the accessory cavities in the anterior and middle fossae have been removed, and looking from before backwards is seen in situ the mucosa of the frontalis, the ethmoid cells and the sphenoid sinuses in bladder-like form, also the root of the cranial nerves. The sphenoid sinuses show an asymmetric development. The left sphenoid sinus extends backwards towards the right and the right sphenoid sinus in front extends towards the left. The sphenoid sinuses touch medially and underneath the corresponding optic nerves.

15. The sphenoid sinuses constitute on both sides the medial wall of the canalis opticus, and the wall of the corresponding third of the sulcus opticus. Figure 75 of the Atlas illustrates this relation by a frontal section. Anterior to the sphenoid sinus the optic nerves are seen entering the canalis opticus. Figure 16 illustrates the same relation by a frontal section. The extremely thin partition wall is seen. The sphenoid sinus on both sides is 17 mm. wide, 22 mm. high, on the right 22 mm. long and on the left 25 mm. long. Figure 17 illustrates by a frontal section the optic nerves on both sides bounded by the lateral wall of the sphenoid sinus, and on the right bordered by the inferior wall of the posterior ethmoid cell.

16. The sphenoid sinuses on both sides constitute the inferior wall of the canalis opticus, and the wall of the sulcus opticus.

Figure 18 illustrates this relation by a frontal section, with this difference, the left sphenoid sinus shows a greater development and it constitutes the wall of the left and middle third of the sulcus opticus.

17. The sphenoid sinuses constitute on both sides the wall of the sulcus opticus.

18. The right sphenoid sinus constitutes the inferior and medial wall of the right canalis opticus, and the wall of the sulcus opticus; the left sphenoid sinus constitutes the inferior wall of the left canalis opticus.

GROUP V.

The relation of the sphenoid sinuses to the optic nerve show seven forms, of which four are on the same side, two on the opposite, and one in the center.

19. The sphenoid sinus on the left side constitutes the inferior wall of the left canalis opticus, and the wall of the entire sulcus opticus.

20. The sphenoid sinus of the left side constitutes the inferior wall of the left canalis, and the wall of the left third of the sulcus opticus. This relation may also occur on the right side.

21. The right sphenoid sinus constitutes the inferior wall of the canalis opticus.

Figure 19 illustrates this relation, a formol preparation of which the bony walls of the right orbit have been removed; the individual accessory cavities and the optic nerve in situ are seen and the above mentioned relation of the optic nerve to the sphenoid sinus is also visible.

Fig. 20, a formol preparation, shows the intimate relation of the left optic nerve to the left sphenoid sinus, which, 12 mm. long, constitutes by an extremely thin bony stratum, the inferior wall of the canalis opticus. The chiasma and the right optic nerve are separated from the sphenoid sinus by sponge-like bones.

22. The right sphenoid sinus constitutes the medial wall of the right canalis opticus. Fig. 21 illustrates this relation by a sagittal section. The thin joint partition wall between the canalis opticus and the sphenoid sinus has been removed, and

the optic nerve is seen taking its course, 5 mm. in length, along the lateral wall of the sphenoid sinus.

23. The sphenoid sinus of the right side constitutes the medial and inferior wall of both canales optici, and the entire wall of the sulcus opticus. Figures 22 and 23 illustrate this relation.

Figure 22, a formol preparation, shows from above the close relation of the right sphenoid sinus to both optic nerves, while Fig. 23 illustrates by a corresponding section the extraordinarily great asymmetry of the sphenoid sinus; the left sphenoid is small, and pushed completely aside.

24. Figure 24 is a preparation illustrating a frontal section; the right sphenoid sinus shows a close relation only to the left optic nerve, which is bordered medially by the extremely thin wall of the right sphenoid sinus.

25. The sphenoid sinus of the left side constitutes the wall of the middle third of the sulcus opticus. Figure 25 illustrates this morphologic relation by a frontal section, showing the left sphenoid sinus in the median line above the nasal septum.

GROUP VI.

The sphenoid sinus and the posterior ethmoid cell on the same side show a close relation to the optic nerves in two forms. One form affects the optic nerve of the same side, the other both optic nerves.

26. The last posterior ethmoid cell of the left side constitutes the medial wall, and the left sphenoid sinus, the inferior wall of the left canalis opticus.

Figure 26 illustrates this relation by a formol preparation. The nasal cavity has been opened from without, the last posterior ethmoid cell, marked by a cross, is seen; also its relation and that of the sphenoid sinus to the optic nerve of the same side.

27. The left posterior ethmoid cell forms the medial wall of the left canalis opticus, and the left sphenoid sinus the inferior wall of the right canalis opticus, also the wall of the right and middle third of the sulcus opticus.

Figure 27 illustrates this relation by a formol preparation of which the inner wall of the orbital cavity has been removed; the mucosa of the accessory cavities is visible. The preparation also illustrates the relation of the left posterior ethmoid cell (marked by a cross), to the left optic nerve, and of the left sphenoid sinus to the right optic nerve. The same relation may exist on the right, also the left sphenoid sinus may be absent.

GROUP VII.

The last ethmoid cells and the sphenoid sinuses on both sides show relations to the optic nerves in one form.

28. The last posterior ethmoid cells constitute on both sides the medial wall of the canalis opticus, and the sphenoid sinuses on both sides the walls of the sulcus opticus.

GROUP VIII.

The last posterior ethmoid cells on both sides, and the sphenoid sinus on one side, show a relation to the optic nerves in two forms.

29. The right posterior ethmoid cell constitutes the inferior and medial wall of the right canalis opticus, and the wall of the right third of the sulcus opticus; the left posterior ethmoid cell constitutes the medial and inferior wall of the left canalis opticus, also the wall of the left third of the sulcus opticus; the right sphenoid sinus constitutes the middle third of the sulcus opticus. This relation is illustrated by figure 67 in the Atlas; it is a frontal section, where in the median line the right sphenoid sinus is visible above the nasal septum. This spur of the sphenoid sinus extends from the ostium sphenoidale 15 mm. to the front, and stands in relation to the middle third of the sulcus opticus and the chiasma.

30. The left sphenoid sinus constitutes the wall of the left third of the sulcus opticus; the left posterior ethmoid cell constitutes the medial and inferior wall of the canalis opticus; the right posterior ethmoid cell constitutes the medial, anterior, lateral and inferior wall of the right canalis opticus, and the wall of the right third of the sulcus opticus.

GROUP IX.

The sphenoid sinuses on both sides and the posterior ethmoid cell on one side, in their bearing on the optic nerves show one form.

31. The left sphenoid sinus constitutes the medial wall of the left canalis opticus; the right sphenoid sinus constitutes the medial wall of the right canalis opticus; the right posterior ethmoid cell constitutes the anterior wall of the right canalis opticus.

GROUP X.

The sphenoid sinus on one side, and the last posterior ethmoid cell on one side, in their relation to the optic nerves show four morphologic relations.

32. The left sphenoid constitutes the medial and inferior wall of the left canalis opticus and the wall of the entire sulcus opticus, also a small part of the inferior and medial wall of the right canalis opticus, whereas the right posterior cell forms the greater part.

33. The left posterior ethmoid cell constitutes the medial, anterior, lateral and inferior wall of the left canalis opticus; the right sphenoid sinus constitutes the medial, anterior, lateral and inferior wall of the right canalis opticus.

Figures 28 and 72 of the Atlas illustrate this relation by corresponding frontal sections. Both instructively show the relation of the optic nerve and the canalis opticus on the one side to the sphenoid sinus, and on the other side to the posterior ethmoid cell; furthermore, the course of the canalis opticus in these cavities, 12 mm. long. The posterior ethmoid cell is 28 mm. long, 30 mm. wide, 26 mm. high; the sphenoid sinuses show an extraordinary asymmetry, the sphenoid sinus on the left side is 30 mm. wide, 22 mm. high, 41 mm. long, on the right side 20 mm. wide, 22 mm. high, and 39 mm. long. The anterior end of the left sphenoid sinus lies asymmetrically in the median line between the lamellae of the septum of the nose.

34. The left sphenoid sinus forms the medial and inferior wall of the left canalis opticus, and the wall of the left and middle third of the sulcus opticus; the right posterior ethmoid cell constitutes the medial and in part the anterior wall of the right canalis opticus.

35. The left posterior ethmoid cell constitutes the medial and inferior wall of the left canalis opticus, the wall of the entire sulcus opticus, and the inferior wall of the right canalis opticus, the right posterior ethmoid cell constitutes the medial wall of the right canalis opticus.

GROUP XI.

The frontal sinus shows two morphologic relations to the optic nerve on one side.

36. The frontal on the same side constitutes the superior wall of the canalis opticus.

37. In the second group, that case was mentioned and described in which the cavity representing the left ethmoid cell merges with the left frontal, and the common cavity constitutes the medial wall of the left canalis opticus and the wall of the left third of the sulcus opticus.

Figures 97 and 98 of the Atlas illustrate this relation by a cross-section.

GROUP XII.

The relation of the turbinate cell of the superior turbinate on one side to the optic nerve shows one form.

38. The turbinate cell, so-called bone-cyst, of the left superior turbinate constitutes the inferior wall of the left canalis opticus and the wall of the left third of the sulcus opticus. This relation is illustrated in frontal section by figure 29. This turbinate cell is 23 mm. long, 19 mm. wide, and 13 mm. in height.

Following the description and illustration of the thirty-eight morphologic relations, which have been arranged in twelve principal groups, we will now discuss certain anatomic facts which are of practical importance and which are to be considered in an etiologic relation. First, the bony walls of the canalis opticus, and of the sulcus opticus; second, the dehiscence of the wall of the accessory cavities; third, the semicanalis ethmoidalis; fourth, the common partition walls of the individual accessory cavities, and fifth, the turbinate cell, so-called bone-cyst, play an important part in diseases of the accessory cavities which cause impaired sight and blindness, Each of these morphologic relations will be separately discussed.

I. THE BONY WALLS OF THE CANALIS OPTICUS AND OF THE SULCUS OPTICUS.

The strength and thickness of those bony walls of the accessory cavities which border the optic nerve, may, in the region of the optic nerve, aid, make difficult or hinder the destruction, impaired circulation, pressure, fracture, and the transmission of the disease. Berger and Tyrmann[4] first examined the differences in the partition wall between the optic nerve canal and sphenoid sinus. They made frontal sections in the plane of the foramina optica, and showed by three illustrations the varying strength and thickness of the bony wall. The first figure illustrates a case in which the partition walls between the optic nerve canal and the sphenoid sinus on both sides are unusually thin. The second illustration shows a preparation of which the spongy partition wall between the optic nerve canal and the sphenoid sinus on both sides is 4-6 mm. thick. The third figure illustrates a case in which the partition wall on one side is thin as paper, on the other it has a thickness of 7 mm. Our observations have shown that the wall between the last ethmoid cell and the canalis opticus and the sulcus opticus is nearly always as thin as tissue paper; in one case it was 1 mm., in another 2 mm. thick. This extremely thin wall between the last ethmoid cell and the optic nerve, figure 30 illustrates by a sagittal section. We have often found a thicker section between the sphenoid sinus and the canalis opticus and the sulcus opticus, the thickness varying between 1, 2, 3, 4, 5, 6, 9 and 12 mm. Figures 31-77 of the Atlas illustrate the extremest differences. Figure 31 illustrates by a frontal section the thin septum between the sphenoid sinus and the optic nerve. Figure 77 illustrates by a frontal section the thickest wall we have observed between the optic nerve, canalis opticus, sulcus opticus and the sphenoid sinus. Figure 32 shows in the region of the optic nerves, a bony wall 6 mm. in thickness, which separates the optic nerve from the sphenoid sinus, and Figure 76 of the Atlas illustrates a frontal section, where bone substance 8 mm. in thickness, separates the chiasma from the sphenoid sinus. In those cases in which the canalis opticus takes its course in varying lengths into the sphenoid sinus or the last ethmoid cell, it constitutes a thin bony wall. Figure 9 illustrates a preparation in which the canalis opticus makes its way, 12 mm. in length, into the last ethmoid cell, which borders it above, below and medially. Figure 28, in the text, and figure 72 of the Atlas, show the course of the optic nerve into the cavities and the thin wall of the canalis opticus. Figure 28, a frontal section, shows on the right side the optic nerve and the canalis opticus in the sphenoid sinus; and on the left side the optic nerve and the canalis opticus in the last ethmoid cell. The canalis opticus, takes its course into the cavities for 12 mm. on the right bordered by the sphenoid sinus, on the left, above, below, medially and laterally by the last ethmoid cell.

Figure 10 illustrates by a sagittal section the optic nerve and the canalis opticus in the last ethmoid cell. Completely bordered by the last ethmoid cell, as is the optic nerve, the canalis opticus, 10 mm. in length, takes its course into the same.

II. THE DEHISCENCE OF THE WALLS OF THE ACCESSORY CAVITIES.

The practical significance of the bony dehiscence is proven by the fact that they may aid or cause orbital and intracranial disturbances as well as sight derangements. The diseased mucosa of the accessory cavities may come in direct contact with the periorbita, the dura mater and the optic nerve sheath and in this way the disease is easily transmitted to the parts mentioned. The dehiscences of the vessel grooves may also lead to a diseased condition of the same. The dehiscences may be the consequences of pathologic changes, senile atrophy, trauma; we may also have to do with artefacts and formative anomalies. We have already discussed these conditions in an article.[5] We will now confine ourselves to anomalies of construction and congenital dehiscences. Such dehiscences have not come under our notice. Zuckerkandl[6] mentions four, Merlin[7] two cases. In the cases observed by Zuckerkandl, the dehiscences of the sphenoid sinus were connected by congenital dehiscences of the lamina papyracea of the ethmoid sinus; all four cases showed dehiscences of the orbital wall of the sphenoid sinus, one case having at the same time a dehiscence of the tuber maxillae. In two cases Merlin observed this dehiscence on the inferior orbital wall. Taking the dehiscences of the frontal into consideration, we have observed in part the extreme attenuation of the superior wall of the frontal between the lamellae of the superior orbital wall, the dehiscences being in part lineal, part dot-like in appearances. Congenital dehiscences and vessel openings may occur on the anterior wall of the frontal. Zuckerkandl and Helly,[8] each had a case under his observation in which a furrow taking its course from the medial part of the arcus superciliaris upwards and in part broken, communicated with the frontal. Under our observation a case in vivo showed this congenital dehiscence, a polyp coming through, or penetrating it from the frontal. Helly noticed vessel openings which connected the frontal with the anterior surface of the frontal bone. We have also observed such vessel orifices in the shape of larger or small holes, in the region of the margo supraorbitalis, and the arcus supraorbitalis. To the congenital dehiscences of the frontal belong those rare cases in which dehiscences of the lamina papyracea of the ethmoid bone open the orbital wall of the frontal at the same time. Zuckerkandl observed this dehiscence in three cases, twice on the left, once on the right side. Merlin observed it once on the left side. We saw three cases of this dehiscence, twice on the right, once on the left side. Concerning the dehiscences of the septal walls of the frontal, according to the observations of Denouvilliers and Gosselin,[9] Winkler[10] and Killian,[11] congenital dehiscences in the form of holes or openings of various sizes may occur on the septum interfrontale. We saw one case in which there was an opening 1.5 mm. in circumference, with smooth edges on the septum interfrontale. Concerning the dehiscence of the lamina papyracea of the ethmoid bone, Hyrtl,[12] already mentions it. Zuckerkandl has observed fourteen cases in which the congenital dehiscence appeared nine times on the left side. Merlin found congenital dehiscences three times in the lamina papyracea of the ethmoid bone, twice on the left, once on the right side. In one case five dehiscences of the lamina papyracea connected the ethmoid cells with the orbital, frontal and sphenoid sinus. Eighteen cases of congenital dehiscences of the lamina papyracea of the ethmoid bone have come under our notice, thirteen being on the left and five on the right side.

The sagittal diameter measured between 4 and 28 mm., the vertical between 4 and 8 mm. In consequence of the dehiscence the ethmoid cells communicated with the orbital cavity; in three cases the frontal, which communicated with the ethmoid cells and the orbital cavity, was also opened.

Figure 23 of the Atlas illustrates a preparation with a dehiscence of the lamina papyracea. Concerning dehiscences of physiologic origin of the sphenoid sinus wall, Zuckerkandl observed on the lateral wall small openings, dehiscences, which connected the sphenoid sinuses with the middle skull cavity. In one case Spee[13] noticed a defect of the sulcus caroticus. On a number of skulls he noticed vessel openings, sometimes symmetrically arranged on both sides, directly under the lateral root of the lesser sphenoid wing. In several instances vessel grooves lead to these vessel openings, small, large and long dehiscences occurring. Zuckerkandl and Hajek have observed dehiscences on the septal wall of the sphenoid sinuses. Dehiscences rarely appear on the walls of the canalis opticus:

out of two hundred cases Gallmaerts[14] abserved two; Holmes,[15] in fifty cases, also saw two dehiscences. Of the three hundred cases under our own notice but one showed a dehiscence of the canalis opticus.

III. THE SEMICANALIS ETHMOIDALIS.

I have described a half or semi-canal, the semicanalis ethmoidalis, which in varying lengths takes its course from the foramen ethmoidale anterius along the wall of the frontal, or the orbital cells to the anterior skull cavity. Through this semi-canal the ethmoidal veins take their course free, and protected by mucosa into the above-mentioned cavities. At the respective ends of the semi-canal the mucosa touches the orbital periosteum and the dura mater. In consequence of this connection transmission of the inflammation may take place, the disease progressing in both directions. Circulatory derangements may also take place, the ethmoidal veins which stand in close relation to the dural venous plexus and the plexus ophthalmicus may be subject to a thrombophlebitis, the same being transmitted to the dural and orbital vein plexuses. Besides the formation of the thrombophlebitis, caused by nervous anastomosis (described by Zuckerkandl and Kuhnt), the ethmoidal vein taking its course into the semicanalis ethmoidalis, may lead to a direct thrombophlebitis, and also to orbital and cerebral complications. Generally speaking, the corresponding marginal grooves of the frontal and the ethmoidal constitute normally a very short canal, the canalis ethmoidalis, which has its beginning in the foramen ethmoidale anterius, ending in the anterior skull cavity with an oblong fissure.

Our examinations in eleven cases showed varying lengths of the canalis ethmoidalis; it varied between 5 and 12 mm. Figure 40 in our Atlas shows by a sagittal section the semicanalis ethmoidalis in an orbital cell. We have designated as orbital cells, those ethmoid cells, which, spread out in the region of the horizontal part of the frontal. One or two orbital cells may occur which open into the middle or upper part of the nasal cavity. The sagittal section of this figure shows the entire course of the semicanalis in a large orbital cell. Inside, it connects the orbital cell with the anterior skull cavity, outside, with the orbital cavity. The foramen ethmoidale anterius extends into the semi-canal, the semicanalis ethmoidalis, arched-shaped on the posterior and medial wall of the orbital cell, 10 mm. long, and leading to an oblong fissure which opens on the anterior canal cavity. We have observed the semicanalis ethmoidalis three times in the frontal; its length varied from 5 to 8 mm.

In the first orbital cell it appeared nine times, its length was from 7 to 10 mm. In the second orbital cell the semicanalis ethmoidalis appeared four times, from 4 to 10 mm. long. In one case the semicanalis ethmoidalis showed a dehiscence between the orbital cells, thereby causing communication between them. Two cases showed a congenital dehiscence of the lamina papyracea, and the presence of the 8-10 mm. long semicanalis ethmoidalis in the orbital cells. Figure 97 of the Atlas illustrates a case in which the ethmoid cells formed a cavity, which merged into the frontal. The foramen ethmoidale anterius was 23 mm. from the anterior wall of the frontal, and took a downward course along the lateral wall of the frontal as a semicanalis ethmoidalis 16 mm. in length.

IV. THE COMMON SEPTA OF THE INDIVIDUAL ACCESSORY CAVITIES.

In view of our personal observations, we now wish to discuss the thin, in fact extraordinarily thin walls of the septa of the individual accessory cavities; they are of practical importance because of the tendency to the transmission of diseases and to perforations. The maxillary sinus may have a common partition wall with the sphenoid sinus and the ethmoid cells. I have proved that the maxillary sinus and the sphenoid sinus may be so enlarged that they lie next to one another. Figure 90 of the Atlas shows this relation by a cross-section. The maxillary sinus is 42 mm. long, 36 mm. wide, the sphenoid sinus 37 mm. to 47 mm. wide and 35 mm. long. The thin partition wall between the cavities is enlarged to a size of 10 mm., and at the same time constitutes the pars maxillaris of the anterior wall of the sphenoid sinus. We have observed six cases of this common partition wall. Its width, i. e., the pars maxillaris of the anterior wall of the sphenoid sinus, measured 4 mm. twice, 6 mm. once, 8 mm. once, and 9 mm. twice. This thin common septal wall between the maxillary sinus and the sphenoid sinus, figure 49 of the Atlas illustrates by a frontal section. Thin, common septa exist between the maxillary sinus and the ethmoid cells. Figure 63 of the Atlas illustrates by a frontal section on both sides the common septa between the maxillary sinus and the last ethmoid cell. An extremely thin partition wall between the maxillary sinus and the last ethmoid cell is illustrated by a frontal section—figure 80 of the Atlas. Figure 96 of the Atlas illustrates by a cross section a preparation, in which the last ethmoid cells on both sides are separated from the maxillary sinus by a common septum. The frontal has common partition walls with the ethmoid cells and the sphenoid sinus. The septa between both frontals, the septum interfrontale, may be sagittal in the median line, or in cases of asymmetry, it may have an oblique direction. Figure 27 of the Atlas illustrates by a sagittal section the asymmetry of the frontals; the left frontal covers the right frontal to the extent of 15 mm., and also extends 30 mm. further upwards. Besides the sagittal septum interfrontale a common horizontal partition wall also exists.

It should be noticed that the frontal on both or only on one side may be lacking, and in consequence there could be no question of a septum wall. The common septa between the most anterior ethmoid cell and the frontal, figure 10 of the Atlas illustrates by a sagittal section. Figures 28, 58 of the Atlas show the common septa between the ethmoid cell (spreading out in the region of the frontal), the bulla frontalis, and the frontal. Figure 28 illustrates a sagittal section in which the thin septum is easy to see between a bulla frontalis 17 mm. long, 22 mm. high, 21 mm. wide, and the frontal sinus 25 mm. long, 6 mm. high, and 34 mm. wide. Figure 58 illustrates by a frontal section the common septum on both sides between the bulla frontalis and the frontal. On the right the frontal is 20 mm. long, 30 mm. wide, 11 mm. high; on the left it is 14 mm. long, 30 mm. wide and 11 mm. high; on the right the bulla frontalis is 20 mm. long, 13 mm. wide, 20 mm. high; on the left it is 20 mm. long, 13 mm. wide, 20 mm. high. The common septum between the orbital cells and the frontal is illustrated by the following sagittal sections. Figure 41 of the Atlas shows the thin arched partition wall between the frontal, which is 23 mm. high, 30 mm. wide, 13 mm. long, and the orbital cell, 27 mm. long, 19 mm. high and 29 mm. wide. Figure 36 of the Atlas shows in a sagittal section the existing communication between the frontal and the orbital cell. Figure 30 of the Atlas illustrates in a sagittal section the extraordinarily thin common septum between the frontal, 19 mm. long, 35 mm. high, 32 mm. wide, and the orbital cell, which is 25 mm. long, 28 mm. wide and 13 mm. high. Figure 26 of the Atlas illustrates a preparation in which the frontal is completely separated from the last ethmoid cell by a common septum. Figure 7 of the Atlas shows the absence of the septum and the merging together of the frontal with the ethmoid cell space.

Figures 31, 37, 52 and 38 in the Atlas show in sagittal section the common septum between the frontal and both orbital cells, also the communication of the individual cavities. Figure 31 shows a thin partition wall between the frontal and first orbital cell, also a thin partition wall between the first and second orbital cells. Figure 37 shows a perfect partition wall between the first and second orbital cells, and communication between the frontal and first orbital cell. Figure 52 shows a complete septum between the frontal and the first orbital cell, and a communication between both orbital cells. Figure 38 shows the communication between the frontal sinus and both orbital cells. The frontal may extend far backwards; it may even reach the foramen opticum, and this expansion may be separated from the posterior ethmoid cell, as well as from the sphenoid sinus by thin common septa. Figure 65 of my Atlas illustrates in frontal section a preparation, in which the frontal, extending to the rear between the orbital lamellae, forms a common thin wall on the right with the right sphenoid sinus, on the left with the left posterior ethmoid cell. This septum between the frontal and sphenoid sinus, that is the pars frontalis of the anterior sphenoid sinus wall, has a a width of 12 mm. Figure 66 of the Atlas illustrates in frontal section a preparation in which the frontal extends posteriorly in the region of the sphenoid sinus and the optic nerve; a thin septum is visible between the frontal and the posterior ethmoid cell.

We have already discussed the common partition walls between the ethmoid cells, frontal and maxillary sinus; the cross-sections in figures 52 and 67 of the Atlas show the septa between the individual ethmoid cells; it is nearly always thin. The common partition walls found between the posterior ethmoid cell and the sphenoid sinus are still to be discussed. The posterior ethmoid cell may rise shelf-like above the sphenoid sinus, or it may be in a position anterior to the sphenoid sinus; the common thin wall between both cavities may be in a vertical or horizontal position, or in both directions in an oblique position. According to our examinations the width of this common septum, the pars ethmoidalis of the anterior sphenoid sinus wall, may vary from 1 to 18 mm. Figure 67 shows in a cross-section the common partition wall between the posterior ethmoid cell and the sphenoid sinus, also the partition wall between both sphenoid sinuses, in a sagittal position; owing to asymmetry of the sphenoid sinuses the partition wall may have various curvatures and positions, and may be even totally lacking in case there is no sphenoid sinus. Figure 68 of the Atlas shows in a sagittal section the shelf-like position of the posterior ethmoid cell above the sphenoid sinus, and the extremely thin common wall between the cavities. Figure 67 of the Atlas illustrates in a frontal section a rare morphologic relation, namely, the common partition walls of the right sphenoid sinus with both posterior ethmoid cells. Figure 28 of the text also shows in a frontal section the common partition wall of the left posterior ethmoid cell with both sphenoid sinuses.

V. THE TURBINATE CELL, SO-CALLED BONE CYST.

The so-called bone cyst is a structural anomaly[18] known as the turbinate cell. Diseased, these turbinate cells may attain various sizes. They appear in the superior and middle turbinate, and, as the turbinate cell of the superior turbinate, according to our observations, may be in close relation to the optic nerve; it will be seen that the practical value of the turbinate cell is due to pathologic changes and growth, especially through pressure.

In cases under our observation the turbinate cell appeared nine times in the middle and twice in the superior turbinate; the turbinate cells twice opened into the middle meatus, at other times in the superior. Its length varied from 8 to 20 mm., the width from 5 to 14 mm., the height from 7 to 16 mm. Figure 18 of the Atlas shows in a sagittal section a turbinate cell in the middle turbinate, 17 mm. long, 20 mm. high, 14 mm. wide, opening into the superior meatus. Figure 11 of the Atlas shows in sagittal section a turbinate cell in the superior turbinate, which opens into the superior meatus. This turbinate cell is 14 mm. long, 14 mm. high and 9 mm. wide. Figure 29 illustrates in frontal section a turbinate cell in the superior turbinate, 23 mm. long, 19 mm. wide and 13 mm. high, which extends back to the region of the optic nerve.

II. CLINICAL OBSERVATIONS.

It is to be regretted that the knowledge of how the diseases of the accessory cavities cause blindness and sight derangements should not yet be firmly established. Excepting individual microscopic and bacteriologic experiments, which prove the continuity of infection, and its passage through the blood vessels, postmortem findings confirm mechanical lesion by which the tumors of the nasopharynx, the sphenoid sinus, the ethmoid sinus and of the ethmoid bone extend into the region of the optic nerve, destroying it or by pressure causing functional derangements and circulatory changes. The postmortem findings show further traumatic lesion of the optic nerve caused by trauma, fractures, fissures in the region of the sphenoid, also pressure of the optic nerve by hyperostosis of the wing of the sphenoid and contraction of the foramen opticum. The postmortem findings confirm suppurations of the accessory cavities, perforations of the cavity walls, basal meningitis, extra- and intradural abscesses, caries, necrosis, thrombosis of the sinus cavernosus, and of the vena ophthalmica. The postmortem findings are faulty, as it is difficult to prove the results of infection, the causal connection, the microscopic and bacteriologic investigations of the diseased accessory cavities. as well as of the optic nerve are wanting, furthermore the more positive assertions of the relation of the optic nerve to the diseased or healthy accessory cavities are lacking, and lastly the investigations of the venae ethmoidales, vena centralis retinae, and the venous radicals of the diseased accessory cavities.

Having assumed that a pathologico-anatomic foundation must be first created, it is clear that there are available only certain stepping-stones, clinical observations, and problematic assumptions, and therefore, at the present time, we can make use only of observations regarding the clinical theory of sight derangements and blindness, canalicular neuritis and atrophy of the optic nerves. Let our clinical observations be preceded by the following remarks. It is known that oculists have established a large number of causes which bring about neuritis and atrophy of the optic nerve. Granting that the causal connection between diseases of the accessory cavities and sight derangements can, and should no longer be doubted, the question as to the accidental appearance of both diseases may still arise. Both of these diseases may surely exist at the same time. Cases have been observed in which an operation of the accessory cavities did not influence the unfavorable progress of the neuritis, furthermore, after a refusal to operate a suppuration, a neuritis optica healed spontaneously. Other cases have come under our own observation, in which the oculist could only confirm the existence of neuritis or atropia optica, without ability to give the cause, and the rhinologist could find nothing abnormal. Blindness or derangements of sight, due originally to a totally different cause, may still show independent empyemas of the accessory cavities. I wish to emphasize this, because in such cases the accessory cavities can in no way be held accountable, even assuming there is a causal connection. And as it.is a fact that the phenomena of disease may closely follow one another without a real or causal connection, it is well to be strictly critical on the matter. This question, in many cases is difficult, or almost impossible to solve. I have touched upon it because in my opinion false interpretations may be easily made.

We will next give our attention to etiologic causes. Under this head come the mechanical and traumatic lesions of the optic nerve, clearly proven by postmortem findings. The tumors of the accessory cavities may cause, in the region of the optic nerve, direct symptoms of pressure. It is not impossible for the tumor to surround the optic nerve, and the same present a normal appearance; as the cases of Bering[19] and Wicherkiewicz[20] and the findings of Ponfick show; furthermore, cases of sarcoma of the sphenoid bone observed by Schmidt-Rimpler and Onodi,[21] showed that both of the optic nerves remained intact. Through postmortem findings, Virchow[22] and Manz[23] were able to trace the cause of blindness back to arrested development of the skull in earliest youth, to a premature ossification of the skull, to a hyperostosis, and in consequence thereof an irregular contraction of the foramen opticum, and pressure on the optic nerve.

Blindness in cases of bradycephalus was, among others, observed by Enslin[24] and Goldzieher[25]. In two cases of blindness we also observed anosmia; there was therefore not only contraction of the foramen opticum, but also of the openings of the lamina cribrosa.

Traumatic injury of the optic nerves and direct and indirect fracture of the canalis opticus are well known. Holder[26] observed fifty-three cases of fracture of the walls of the canalis opticus. The etiologic significance of these cases was made evident by postmortem findings.

Infection, the transmission of inflammation, bone dehiscences and derangements of circulation are specially to be mentioned as etiologic phenomena of suppuration of the accessory cavities. We have already spoken of the faulty and unsatisfactory postmortem findings; they establish, in general, the infection, the causal connection of the accessory cavities, suppuration with visible bone destruction, thrombophlebitis, meningitis, orbital and cerebral abscesses. The investigations are wanting, of the changes of the septa, the walls of the accessory cavities, and the vessels, particularly those of the ethmoidal veins, and the vena centralis retinae, and of the optic nerve. We have two accurate observations, made by Orman[27] and Hajek,[28] who have established the cause of infection by way of the blood vessels and tissue continuity.

The case of Ortman showed the presence of an empyema of the sphenoid sinuses, an extradural abscess of the sella turcica, and a thrombosis of the sinus cavernosus on both sides. The bone in the region of the sella turcica was a discolored reddish brown; a microscopic examination showed the following:

The presence of numerous diplococci lying between small particles of pus on the mucosa and periosteal plane of the lining of the sphenoid sinuses, also their presence in its substance, infiltrated by hemorrhagic foci.

Sections through the bone (body of the sphenoid) and the membranes showed the presence of diplococci. This finding showed that infection of the meninges may take place by direct continuity. Added to the inflammation and coagulation of the secretions in the sphenoid sinuses, were a periostitis and ostitis purulenta, which, penetrating the bone, caused a periostitis on the cerebral plane, and in that way affected the hard and soft membranes of the brain.

The case observed by Hajek showed the presence of an empyema of the ethmoid labyrinth and meningitis. The section showed a diffuse, fibrous, suppurative, acute pachymeningitis interna, and leptomeningitis of the base and convexity of the brain with acute internal hydrocephalus, inflammation of the left ethmoid cells, edema of the mucosa of the maxillary sinuses. The exudate of the brain showed streptococci pyogenes. Hajek expressed himself after the investigations in the following manner:

"We have to deal with the influx of virulent streptococci into the inflamed mucous membrane tissues, also with an invasion of streptococci into the circulatory system of the blood vessels. In consequence of the familiar vein anastomoses between the mucosa of the ethmoid labyrinth and the dura mater, the meninges were soon infected, but the bone and its marrow substance were not affected."

Westenhoefer's[29] earlier statements concerning the nasal lymphogenic origin of cerebrospinal meningitis has not so far been microscopically confirmed; he recently declared that the lymphogenic origin and the cause of infection must be established by microscopic investigations. My cases, which were subjected to postmortem observation, as well as the collected material are being reviewed from a pathologic standpoint by Dr. B. Entz, assistant in the Pathologico-Anatomic Institute in Budapest. These researches are not yet completed; it was therefore impossible to incorporate them in this article.

For further information on the subject we will make use of our observation and postmortem findings, bearing on the complications of the accessory cavities. We will discuss them quickly (divided into groups) before we go further into the subject of etiologic phenomena.

In cases of maxillary sinus empyema, Foucher[31] notes orbital phelgmon, thrombophlebitis of the plexus pterygoideus and of the plexus ophthalmicus. Mair[32] notes caries of the ethmoid bone, perforations of the lamina cribrosa and abscess of the orbital lobe.

Panas[33] found necrosis of part of the orbital roof, suppurative osteoperiostitis of the orbit, and abscess of the frontal lobe. The case of Dmochovszky[34] showed numerous perforations of the maxillary sinus, rupture into the sphenoid sinus, suppurative meningitis, intradural abscess, abscess of the frontal lobe.

Westermayer's[35] showed rupture into the fossa pterygopalatina, caries, perforation of the large sphenoid wing, abscess of the temporal lobe.

In cases of frontal empyemas the following findings are noted:

In the case of Bousquet[36], perforations of the posterior and inferior frontal wall with abscess of the frontal; in the case of Koehler,[37] perforation of the posterior frontal wall, intradural abscess and meningitis basilaris; in the case of Schindler[38], perforation of the posterior frontal wall, frontal abscess and pyemia; in the case of Zirm[39], thrombophlebitis of the ocular veins and of the sinus cavernosus, orbital abscess, suppurations and putrefactions of venous thrombi; in the case of Huguenin[40], encephalitis and meningitis; of Mueller[41], abscess of the left parietal bone, perforation, communication with an extradural abscess, meningitis on the side of the convexity; Paulsen[42], perforation of the posterior frontal wall of the dura, intradural abscess, meningitis; Huguenin, perforation of the posterior frontal wall, suppurative pachymeningitis and leptomeningitis, intradural abscess, connective tissue adhesion of the membranes with the brain surface; Knapp[43], perforation of the posterior frontal wall, periostitis of the orbital plate of the frontal, the dura adhering or growing to the discolored brain, an incipient frontal lobe abscess; Bourot-Lecard[44], periostitis of the frontal and orbital plates of the frontal, thrombophlebitis of the ocular veins and of the sinus cavernosus, suppurative meningitis; Redtenbacher[45], perforation of the posterior frontal wall, frontal abscess, intradural abscess; Lennox-Browne,[46] perforation of the posterior frontal wall, extradural abscess, pachymeningitis and leptomeningitis; Hoppe[47], congenital defect of the posterior frontal wall, meningitis of the convexity and base of the brain; Krecke[48], extradural abscess, frontal lobe abscess, rupture into the lateral ventricle; Wallenberg[49], perforation of the posterior and inferior frontal wall, intradural abscess; Silex[50], perforation of the posterior and inferior frontal wall, abscess of the brain on both sides; Milligan[51], septic thrombosis, meningitis; E. Fraenkel[52], thrombophlebitis of the sinus longitudinalis superior with pyemia; Macewen-Millar[53], thrombophlebitis of the sinus longitudinalis superior in the frontal region, extradural and intradural abscess, pus coating of the brain surface, pyemia; Roth[54], external frontal abscess, suppurative meningitis, on the right, encephalitis, on the left, abscess; Carver[55], thrombosis of the sinus longitudinalis superior, meningitis of the convexity; Cholle[56], meningitis.

The following are findings in cases of ethmoidal empyema:

In the case of Bigbie[57], perforation of the lamina cribrosa of the ethmoid bone and the frontal, and frontal abscess; Schaffer[58], perforation of the lamina papyracea of the ethmoid, periostitis orbitalis, perforation of the superior wall of the orbital cavity, frontal abscess; Pauncz[59], and Jacubasch[60], abscess of the brain; Ogston[61], Warner[62], Ewald[63], meningitis; Trousseau[64], necrosis of the ethmoid bone and meningitis; Hajek[65], meningitis.

In cases of empyema of the sphenoid sinus the findings are as follows:

Horner[66], blindness and exophthalmus on the right, the section showing caries of the base of the sphenoid bone, and the adjoining parts; Panas[67], blindness, exophthalmus, phlegmona orbitalis in the course of ostitis of the sphenoid bone; Rouge[68], strabismus divergens, loss of the left half of the field of vision, the postmortem findings showing suppurative periostitis and empyema of the left sphenoid sinus; Raymond[69], blindness, exophthalmus of both sides, the postmortem showing basal meningitis, an exudation of pus compressing the chiasma, thrombosis of the sinus cavernosus, and of the venae ophthalmicae, caries and perforation of the sphenoid plate; Ortman, extradural abscess of the sella turcica, thrombosis of both sinus cavernosi, periostitis, and empyema of the sphenoid sinuses; Gruenwald[70], basal meningitis, pus in the sphenoid sinuses and the maxillary sinuses; Vince[71], perforation of the basilar apophysis and meningitis; Thiroloix[72], extradural abscess of the sella turcica, meningitis; Flatau[73], perforation of the posterior wall, meningitis; Zoeckendoerfer[74], meningitis; Pekastovsky[75], necrosis of the sella turcica, meningitis, thrombosis of the sinus longitudinalis; Scholz[76], periostitis suppurativa, perforation of the superior and lateral sphenoid sinus wall, meningitis, arrosion of the sinus cavernosus, thrombosis of the sinus caroticus and petrosus dexter, intradural abscess of the middle cranial fossa; Schlagenhaufer[77], softening of the body of the sphenoid, meningitis and thrombophlebitis of the sinus longitudinalis; Pauncz[78], thrombophlebitis purulenta sinus circularis Ridlei et sinuum petros. inf. et sup.; Halasz[79], empyema of the sphenoid sinus and encephalitis; Schroeder[80], empyema of the sphenoid sinuses, perforation, meningitis, thrombophlebitis of the sinus cavernosus and petrosus.

In the case of combined empyema of the accessory cavities, the following postmortem findings are noted:

Weichselbaum[81], empyema of the right maxillary and frontal cavity, abscess of the upper eyelid, extradural abscess and frontal lobe abscess; Gruenwald, empyema of the maxillary and sphenoid sinuses, periostitis suppurativa, and softening of both posterior cornua; Pauncz[82], empyema of the maxillary sinus and the ethmoid cells on the left, rupture from the maxillary sinus into the orbital cavity, meningitis basilaris, abscess of the brain; Hansberg[83], frontal empyema on the right side, ethmoid empyema on both sides, caries of the ethmoid, meningitis, frontal lobe abscess on the left side; Duplay[84], exophthalmus on the right side, total blindness accompanied by basal meningitis, particularly over the sella turcica, suppurative phlebitis in most of the sinuses, several foci of pus in the orbital cellular tissue, pus in the sphenoid sinuses and in the right ethmoid cells; Russel[85], diminution of sight, ptosis of the upper eyelid, and fixation of the bulb on the left, the postmortem showing intradural abscess of the middle cranial fossa, empyema of the sphenoid sinuses, and of the posterior ethmoid cells, thrombosis of the sinus cavernosus, petrosus and of the left vena ophthalmica; Vossius[86], empyema of the sphenoid sinus, and of the left ethmoid labyrinth, with caries of the sphenoid bone, thrombophlebitis of the vena ophthalmica superior, the vena angularis and vena facialis anterior sinistra, suppurative pachymeningitis and leptomeninigitis; Holmes[87], empyema of the frontals, the ethmoid cells, the sphenoid sinuses, diplopia, neuritis optica on both sides, abscess of the right frontal lobe; Demarquay[88], marked drooping of the eye, the eye fixed, not sensitive to light on the right side, the postmortem showing the sinus cavernosus bathed in pus, the sphenoid, ethmoid, and maxillary sinuses filled with pus; Panse[89], empyema and tubercular granulation in the frontals, ethmoid cells, maxillary and sphenoid sinuses, neuroretinitis on both sides, tuberculosis of the ethmoid bone and anterior portion of the sphenoid, the orbital roofs on both sides carious and covered with a caseous mass, local meningitis, hydrocephalus internus, caseous pneumonia; Leber[90], nasal tumor, empyema of the sphenoid sinus, blindness, atrophy of the optic nerve, suppurative meningitis and thrombophlebitis of the brain sinus; Finlag[91], empyema of the sphenoid sinuses and ethmoid cells, thrombophlebitis of the sinus cavernosus and circularis.

As the postmortem findings have shown us, the complications stand in close relation to the infection. That infection may take its course through tissue continuity and the blood vessels has been proven, that it may make its way through the lymph vessels, along the lymph spaces of the olfactory nerves through the openings of the lamina cribrosa, is possible, but has not been, up to date, microscopically and bacteriologically confirmed. The transmission of the inflammation, the diseased processes of the accessory cavities, the pathologic changes of the contents of the orbit, the canalis opticus, the optic nerve, the intracranial and cerebral complications stand in close relation to the venous system and to other important etiologic conditions. To the circulatory disturbances belong hyperemia, edema of the tissues, the optic nerve and its sheath, hemorrhages, embolisms, thrombophlebitis and thrombosis. We combine as one those connections between the venous radicals and the venous plexus, which are of practical value and which have made clear to us the origin of circulatory disturbances, as well as of thrombophlebitis and thrombosis. Zuckerkandl's investigations have shown that the veins of the nasal mucosa bear a relation to the facial veins, the anterior ethmoidal veins to the dura and pia mater; the venous radicals of the nasal mucosa to those of the pharynx and palate and to the venous plexus of the fossa pterygopalatina. We are familiar with the connection existing between the venous plexus of the nasolachrymal canal and lachrymal sac and the vena facialis anterior, the vena ophthalmica and the vena infraorbitalis.

The vena lacrimo-facialis, described by Zuckerkandl, stands in relation to a larger branch, which has its origin in the anterior ethmoid cells, and which bores its way through the lachrymal bone. The venous plexus of the maxillary sinus bears a relation to a branch which bores its way through the sphenoid sinus wall, and with the vena ophthalmica facialis. According to Kuhnt[92], the venae perforantes of the frontal bring it into a relation with the dural venous plexus. Kuhnt[93] and Gurwitsch[93] found connections between the veins of the frontal, the vena ophthalmica superior and vena supraorbitalis. The vena ethmoidalis anterior and posterior empty into the vena ophthalmica superior. The vena ethmoidalis anterior may be joined with the vena angularis and the vena supraorbitalis. According to Onodi, the vena ethmoidalis anterior may lie bare in the semicanalis ethmoidalis in a course of varying length, and in the frontal and first and second orbital cells. The venae ophthalmicae superiores are connected with the venae ethmoidales, the vena centralis retinae, and also the vena ophthalmica inferior. The veins of the eye are connected with the facial veins, the veins of the nasal cavity, the plexus pterygoideus, as well as the sinus cavernosus. The vena centralis retinae may be directly joined to the sinus cavernosus. The veins of the sphenoid sinuses may stand in direct relation to the plexus ophthalmicus and the sinus cavernosus. According to Kraus[94] it is possible that the vena ophthalmica inferior or a branch may make its way out into the fossa pterygopalatina, furthermore, that the central vein of the opticus may take its way through the inferior fissure. Owing to numerous anastomoses the circulatory disturbances are easily explained. Hyperemia and symptoms of congestion are also explained by reason of these vein connections. The transmission of inflammation to the orbital tissue and to the sheath of the optic nerve may cause lesion and pressure of the vessels, the separate venous radicals may become directly diseased, particularly those surrounded by the inflamed orbital tissue, and the ethmoidal veins, covered by the diseased accessory cavity mucosa, may be subject to thrombophlebitis, and because of the vein connections mentioned may lead to embolisms, as well as to a dissemination and destruction of venous thrombus.

Kuhnt saw three cases of embolism of the vena centralis retinae from maxillary sinus empyema. An operation was refused, so perforce there can be no further information on the subject. The case of Fouchet showed the presence of maxillary sinus empyema, thrombophlebitis of the plexus pterygoideus, and of the plexus ophthalmicus. In cases of empyema of the frontal the following observations were made:

Zirn, thrombophlebitis of the veins of the eye and of the sinus cavernosus. Bourot-Lecard, thrombophlebitis of the veins of the eye and of the sinus cavernosus; E. Fraenkel, Macewen-Millar and Carver, thrombophlebitis of the sinus longitudinalis superior. Raymond, sphenoid empyema, thrombosis of the sinus cavernosus and of the venae ophthalmicae; Ortman, thrombosis of both sinus cavernosi; Pekastovsky, thrombosis of the sinus longitudinalis; Scholz, thrombosis of the sinus caroticus and petrosus dexter; Schlagenhaufer, thrombophlebitis of the sinus longituidinalis; Pauncz, thrombophlebitis purulenta sinus circularis Ridlei et sinuum petros. inf. et sup.; Schroeder, thrombophlebitis of the sinus cavernosus and petrosus.

In cases of combined accessory cavity empyemas the following are the findings:

Duplay, empyema of the ethmoid cells and sphenoid sinus, suppurative phlebitis in most of the sinuses; Russel, empyema of the ethmoid cells and sphenoid sinus, thrombosis of the sinus cavernosus, petrosus, and the left vena ophthalmica; Vossius, empyema of the ethmoid cells and sphenoid sinus, thrombophlebitis of the vena ophthalmica superior, the vena angularis and the vena facialis anterior sinistra; Finlag, empyema of the sphenoid sinuses, and the ethmoid cells, and thrombophlebitis of the sinus cavernosus and circularis.

Concerning transmission of the inflammatory process, the congenital, physiologic and bone dehiscences caused by senile atrophy play an important part. Congenital dehiscences have been discussed in a separate chapter. Owing to the presence of congenital bone dehiscences the mucosa of the frontal and of the maxillary sinus may be in relation with the external bone periosteum and the periorbita; the mucosa of the frontal and the sphenoid sinus may touch the dura mater, the semicanalis ethmoidalis may bring the frontal and the orbital cells outwardly into relation with the periorbita, inwardly with the dura mater. The dehiscences of the canalis opticus, of the ethmoid cells and the sphenoid sinus may connect the mucosa of the cavities with the sheath of the optic nerve. The dehiscence of the lamina papyracea of the ethmoids connects the ethmoid cells with the periorbita. Concerning the dehiscences of senile atrophy, Zuckerkandl and we have observed the extreme attenuation of the walls of the sphenoid sinus, in places with holes and defects of various sizes. In a case of Zuckerkandl, the frontal communicated with the skull cavity, in the case of Merlin, the frontal did the same with the orbital cavity. In such cases periostitis, perineuritis optica and meningitis with its accompanying phenomena often occur. Dehiscences between the accessory cavities and the cranial cavity may bring about pachymeningitis, extradural abscess, rupture and cerebral abscess; dehiscences between the accessory cavities and the frontal, the cheek and fossa pterygopalatina may cause periostitis, phelgmon, abscesses, and thrombophlebitis.

Dehiscences between the accessory cavities and the orbital cavity may cause orbital emphysema, periostitis, orbital phlegmon, orbital abscess and thrombophlebitis; dehiscences of the canalis opticus may cause periostitis, perineuritis optica, neuritis and atrophy of the optic nerves. In addition to these conditions the inflammatory process may be transmitted to the periosteum, the bony walls of the accessory cavities and to the neighboring tissues, causing inflammation of the bony parts and destruction of the bones, also caries, necrosis, perforation of the individual accessory cavities, phelgmon, abscesses of the orbit and of the adjoining parts of the individual accessory cavities, accompanied by all the phenomena of disease. If the inflammation is transmitted through the bones, it may easily affect the optic nerve and its sheath in the region of the canalis opticus, invading the orbit by way of bone inflamation; periostitis appears, which transmits itself directly to the canalis opticus, causing diseased optic nerves, also orbital phlegmona, orbital abscesses, as well as pressure on the optic nerves and its vessels; furthermore, thrombophlebitis of the veins of the eyes, and its serious accompanying phenomena. Communication between the frontals and the ethmoid cells, the so-called orbital cells, may lead to a direct infection of these cavities. The thin common partition walls of the individual accessory cavities may, by means of a process of perforation, result in invasion of the neighboring accessory cavities. A rupture may therefore connect the frontals, as well as the sphenoid sinuses. In this way the diseased process may be directly transmitted from the maxillary sinus to the ethmoid cells, and to the sphenoid sinus, and continued on the same side from the ethmoid cell to the sphenoid sinus. In case of a rupture of the common wall of the ethmoid cell, the contralateral sphenoid sinus may also become diseased. These conditions may also run their course in the opposite direction. We will now discuss a few complications in connection with accessory cavities of the empyema, which not yet have been mentioned.

In case of maxillary sinus empyema, the following complications are noted: Epiphora, blepharospasmus, reflex neurosis, asthma, angina pectoris, dacryocystitis purulenta, fistulous condition of the tear-sac. In the case of Nolthenius[95] there was rupture of the maxillary sinus suppurations in the direction of the nasolachrymal canal. In the case of Treitel[96] granulations and sequestrations of the inner wall of the maxillary sinus caused blenorrhea of the lachrymal sac and a fistulous growth. Asthenopic conditions have been observed by Gruenwald, Caldwell[96] and others. Ziem and Fromaget saw a case that was cured by operation; Kuhnt, an improvement, after treatment of iritis; Ziem and Kuhnt, improvement in sight in a case of cataract. Kuhnt saw the appearance of neuroretinitis. Grohman[98] observed a hyperemia of the optic nerve papilla; Depaques[99] observed an obliteration of the borders of the optic nerves, with impaired sight; Courtaix and Kolarowitsch report amblyopias and amauroses of dental origin. In the cases of Koerner[100], LeFort[101], Pagenstecher[102], there was exophthalmus from edema of the retrobulbar cellular tissue, also impairment of sight to the extent of blindness, from pressure on the optic nerves and its vessels. There was a transient amblyopia or amaurosis in the cases of Brueck[103], Pasquier[104], Buzer [105], Galezowski[106] and Onodi [107].

The diseased condition of the bone leads to rupture, to fistulae and abscesses. Abscesses and fistulae of the hard palate were observed in the cases of Gruenwald[108] and Panzer[109], phlegmon of the fossa pterygopalatina in the cases of Tedenat[110], also Westermayer[111]. Rupture of the medial wall of the maxillary sinus into the inferior meatus was observed by Bournonville[112], Jansen[113], Flatau[114], and Lichtwitz[115]; into the middle meatus by Killian[116]. In the case of Dmochowski[117] a rupture of the maxillary sinus into the sphenoid sinus took place. Rupture of the superior wall of the maxillary sinus, that is, the inferior wall of the orbital cavity, was observed in the cases of Bauby[118], Dmochowski, Kuhnt, Salva[119] and Fisher[120]. Orbital phlegmona and orbital abscesses may appear without the presence of rupture, and as we have observed in the quoted cases, may lead to intracranial suppurations and to thrombophlebitis of the venous radicals and of the sinuses.

In cases of frontal empyema the following complications may arise: Reflex neurosis, bulbar, periorbital neuralgia and rarely lachrymal blenorrhea. According to Kuhnt and Lichtwitz, hyperemia of the papilla, filling of the veins and peripapillar retinal infiltration may exist. Kuhnt, Onodi and others noted exophthalmus and diplopia. Schmiegelow[120] and

Jacqueau[121] saw acute exophthalmus. Kuhnt reported opacities of the lens. According to Lapersonne[122] transitory or permanent paralysis of the nerves of the eye muscle, edema of the retrobulbar connective tissue, orbital phlegmon, orbital abscess, periosteal inflammations and rupture of the bony walls have often been observed. Bourot, Lecard, Carver and Schroeder have observed periostitis and subperiosteal abscess of the superior orbital wall, and at the same time caries, necrosis, sequestra. Spencer-Watson[123] removed two sequestra of the orbital lamina of the frontal and Panas[124] one. We do not so often see changes on the anterior frontal wall; periostitis, phelgmon of the frontal derma and of the upper eyelid and abscesses have been observed by Baeumler[125], Steinthal[126], Koehler[127], Roth and MacEwen-Millar, perforation by Botey[128]. Perforations of the posterior frontal wall, orbital abscess, thrombophlebitis, intracranial and cerebral complications have been mentioned in the reported cases.

In cases of ethmoid empyemas, Burger[129], Caldwell[130], Hajek[131], and Gruenwald observed asthenopic difficulties. Cases of retrobulbar neuritis and congestion neuritis are known; they will be discussed later. Dacryocystitis purulenta, orbital emphysema. Displacement of the eye-ball may be caused by mucocele of the ethmoid cells, as well as by ectasia of the empyema. It is a known fact that suppurative swelling of the anterior ethmoid cells may cause displacement of the eye-ball.

Exophthalmus without impaired sight in cases of ethmoid mucocele was observed by Hjorth[132] and Onodi[133], exophthalmus with diplopia by Baurowitz[134], exophthalmus, neuritis optica by Mann[135], exophthalmus and blindness by Richet, seropurulent contents of the mucocele with displacement of the eye-ball by Schmiegelow[136]; intermittent exophthalmus, Eversbusch[137]. Orbital phlegmon, orbital abscess, perforation of the ethmoid are also noted, and cases with intracranial and cerebral complications have been reported.

In cases of sphenoid sinus empyema, the following observations are noted: Cases of congestion of the papilla, retrobulbar neuritis, and atrophy of the optic nerve will be more fully discussed later. Orbital phlegmona, exophthalmus and diplopia may occur. Baumgarten[138] reports a case of oculomotor paralysis and Post[139] a cured case of necrosis of the small wing of sphenoid with exophthalmus and permanent amaurosis.

Cases with intracranial complications have already been mentioned.

Cases of intracranial and cerebral complications with combined empyema of the accessory cavity have also been reported. Cases of neuritis and atrophy of the optic nerves will be more fully discussed later.

In the case of Sokos and Luc[140], diplopia occurred in empyema of the maxillary and frontal sinus. In a case of maxillary and sphenoid sinus empyema, Lapersonne observed a complete oculomotor paralysis. Ingersoll observed exophthalmus in a case of empyema of the frontal and of the ethmoid cells, and Broeckert[141] observed dimness of the vitreous body and detachment of the retina in a case of empyema of the frontal and ethmoid cells. Hoffman[142], in empyema of the frontal, the ethmoid cells and of the sphenoid sinus, found rupture into the orbit and orbital abscess, and in a case of empyema of the ethmoid cells and the sphenoid sinus exophthalmus and diplopia. F. R. Alexander[143] saw, in connection with an empyema of the sphenoid sinus and the ethmoid cells, a metastasis of the eye, and abscess of the vitreous body.

We now come to the question of atrophy of the optic nerves and canalicular neuritis brought about by diseases of the accessory cavities. As we have already mentioned, starting from the orbit the inflammation may be directly transmitted to the optic nerves, also pressure on that portion without vessels, and upon the vessels of the optic nerves may lead to impaired sight and blindness. Starting from those accessory cavities which are in close relation to the optic nerve, the inflammatory process may be directly transmitted to the walls of the canalis opticus and the sheath of the optic nerve, and that this transmission is furthered by the presence of possible bony dehiscences, is self-evident. In those cases in which the common septal wall between the canalis opticus and the accessory cavities is very thin, periostitis and disease of the bones may lead directly to perineuritis and its subsequent symptoms.

It has been possible to explain the circulatory derangements on the score of the vein connections and origin in the sinus which have been described. The postmortem findings quoted from Duplay, Horner, Panas, Rouge, Russel, Raymond.

Demarquay, Vossius, Holmes and others confirm the causal connection of sight derangement and blindness with diseases of the ethmoid cells and sphenoid sinuses. It is unfortunate that we lack the confirmation of the course of infection, the investigation of the optic nerve, its sheath, the optic vessels, the proof of the more intimate relations existing between the individual accessory cavities and the canalis opticus and between the sulcus opticus and the optic nerve and chiasma, with their possible changes. A number of observations are placed at our disposal, which lean or point towards a causal connection with positive cures of the optic nerve in connection with unfavorable course and also negative results in spite of operations.

We are acquainted with several cases in which, in spite of the presence of suppuration and destruction of the bony walls of the sphenoid sinus, the sight was not impaired. Berger and Tyrmann mention the slow erosion or wearing away of separate parts of the sphenoid body, though no derangement of sight took place, but later meningitis appeared. Baratoux[144] mentions a case in which a large part of the sphenoid body was thrown off through the nose, causing no disturbance whatever. Hajek saw a number of cases of syphilitic destruction of the anterior wall of the sphenoid sinus without special symptoms of any other kind. Flatau[145] reports twenty-six cases of empyema and caries of the sphenoid sinus, but mentions no derangement of sight. Foucher[146] describes the case of a girl of fifteen with necrosis of the turbinates and the sphenoid sinuses; antiluetic treatment was instituted, bone sequestra were formed and the girl died; sight disturbances were not observed. The observations of Schaffer,[147] Onodi,[148] Schmiegelow and Hoffman[149] also show that in many cases of diseased sphenoid sinuses no changes in the functions of the optic nerves took place. Hinkel has treated twenty cases of sphenoid sinus empyema without noticing changes in the field of vision or that they brought about diseases of the fundus of the eye. In contrast with the observations of Grünwald, Ziem,[150] Berger,[151] Kuhnt and Bryan,[152] Henrici and Haffner,[153] in thirty-six cases of accessory cavities diseases found normal field of vision. According to our opinion, shared by Hoffman, these facts are to be explained by reason of the observations and conditions which we have discussed.

We have spoken of those cases in which the canalis opticus and the optic nerve stand in no close relation to the ethmoid cells and the sphenoid sinuses. Furthermore, we have mentioned the observations and illustrated those preparations which show the different thicknesses of the bony substance between the sphenoid sinus and the optic nerve.

In the transmission of the inflammatory process of the sphenoid sinus, two facts play a protective and restraining part. First, we have seen how far removed the sphenoid sinus may be from the region of the optic nerve, and second, the bone substance, varying in thickness from 1 mm. to 12 mm., which separates the sphenoid sinus from the optic nerve. These facts show that empyema may cause destructive processes of the bones without sight derangements resulting.

It would lead too far afield to speak of the different causes of neuritis; we will therefore confine ourselves solely to the diseased accessory cavities in the region of the optic nerve. In this connection I must call attention to the almost mechanical theory which sought to bring about a connection between canalicular diseases of the optic nerve with diseases of the sphenoid sinus.

By reason of my investigations I have taken issue with this theory and have proven[154] that in many cases the sphenoid sinus has nothing at all to do with the optic nerve, and intimate relations exist only with the last posterior ethmoid cell. These facts have already been considered by Schmiegelow, Alexander and Hoffman. Pauncz,[155] who speaks of these facts regarding the section of the optic nerve in the canalis opticus says further that the intracranial part of the optic nerve, between the canalis opticus and chiasma, "stands almost solely in close relation with the sphenoid sinus only." This statement has been refuted by the above given findings, also his remark "that this section of the optic nerve is always separated from the sphenoid sinus by a thick bony wall." I have mentioned in my works and on this occasion must again emphasize the fact that when the ethmoid cells and the sphenoid sinus become diseased simultaneously, both cavities are to be taken into consideration; under no condition is it to be supposed that the sphenoid sinus alone is the foundation for the causal connection. Schmiegelow is correct when he brings sight derangements into causal connection with the ethmoid cells and the sphenoid sinus, not making one sinus or the other responsible. On the contrary, Halstead[156] and Pollatscheck[157] in empyema of the ethmoid cells and of the sphenoid sinus, trace back the derangement of sight solely to a diseased sphenoid sinus, without establishing the elimination of the ethmoid cells, whereas the assumption of a causal connection with empyema of the ethmoid cells might as well have been made. According to our morphologic findings, transmission of the inflammation from the posterior ethmoid cells as well as from the sphenoid sinus, to the optic nerve, is possible, as well as circulatory disturbances.

Mendel[158] and Lapersonne have stated that the appearance of neuritis on one side is usually characteristic of nasal origin. Mendel finds "that almost half the cases of one-sided optic nerve inflammation are in a large measure of nasal origin." He says that unilateral choked disc is mostly an orbital affection; on both sides it is an intracranial affection.

Lapersonne emphasizes the characteristics of one-sided disease: "Optic neuritis is rarely observed in frontal sinusitis, somewhat more frequently in maxillary or ethmoid empyema, but it is especially a manifestation of sphenoid sinusitis. One main characteristic of this neuritis when purely of sinus origin is that it is unilateral. Although both nerves may be attacked in the canal by a double sphenoid sinusitis, a double edematous neuritis should make us look for an intracranial cause."

Prof. H. Sattler[159] in answer to my question expressed himself in the following manner: One-sided neuritis optica, or atrophy of the optic nerve, is in no way characteristic of diseases of the sphenoid and ethmoid sinuses; it may be induced by inflammation, hemorrhages, tumors, at the cerebral end of the canalis opticus and by toxic phenomena.

The report submitted by Mendel and Lapersonne cannot hold good; speaking against the assertions above given, there are, in the first place, those cases in which diminution of sight on both sides was observed in one-sided disease, furthermore the morphologic findings discovered and described by us establish the possibility of the lesion of both optic nerves and chiasm in diseases of one posterior ethmoid cell or a sphenoid sinus on one side. One fact presented by Mendel and Lapersonne we are able to confirm, namely, that in most cases heretofore under observation, a unilateral disease of the accessory cavities brought about the sight derangements and blindness. The number of observed cases of suppuration of the accessory cavities on both sides is considerably smaller, as well as the number of cases of contralateral and double-sided sight derangements in one-sided diseases of the accessory cavities.

Before considering these cases collectively, we wish to call attention to an etiologic point and this is the artificial impairment of sight caused by an operation. This occurred in a case observed and published by Hirschberg[160] in which, after operation for empyema of the right maxillary, ethmoid and sphenoid sinuses, a superior, partial immobility of the pupil ensued. This traumatic lesion of the optic nerve was brought about by the clearing out of the posterior ethmoid cells and the sphenoid sinuses through the maxillary. The topographic relations discussed explain these artificial injuries, particularly the varying course of the optic nerve along the posterior ethmoid cell and the sphenoid sinus. A glance at figures 8, 9 and 27 will clearly explain the origin of a traumatic lesion of the optic nerve in cases of cleansing out of the cavities in question.

Concerning disease of the accessory cavities on one side with unilateral sight derangements and blindness of the same side, we will mention the cases already noted with postmortem findings of Duplay, Horner, Panas, Rouge, Russell, Raymond, Demarquay, Leber and Holmes. Cases are mentioned in which accurate data concerning nose-findings, diagnosis and operative measures are lacking. Berger reports two cases of neuritis ending with amaurosis. Grossman[161] thought that in four cases there was an etiologic connection between sight impairment and the diseased sphenoid sinus. Snellen[162] traced two cases of optic nerve atrophy back to sphenoid sinus empyemas. Knapp[163] assumes that in one case a sphenoid sinus empyema led to changes of the papilla.

Hirschman writes me of a case in which neither a necropsy nor an operation was permitted and as the cause of a temporal hemianopsia, the atrophic discoloration of the temporal half of the left papilla and the paleness of the same half on the right side, assumes a diseased sphenoid sinus or a tumor in the region of the sella turcica.

In the cases of Lapersonne and Gronbäk[164] the operation was not successful. In the case of Gronbäk the amaurosis with empyema of the ethmoid cells remained unchanged. Operations in Lapersonne's three cases did not influence the unfavorable progress of the neuritis optica. In the case of Post, necrosis of the smaller wing of the sphenoid caused exophthalmus and amaurosis. After the extraction of the necrotic bone the eye resumed its normal position, the amaurosis, however, remained. In the case of Risley[165] empyema of the ethmoid cell and the sphenoid sinus caused blindness of the eye.

We will now discuss those cases in which sight derangements on one side with diseased accessory cavities were observed and cured. But we must touch first on the case of Coppez and Lor,[166] who observed in a girl 20 years old, a neuritis optica of one side which was cured in spite of the fact that the girl would allow no surgical operation of her chronic sphenoid sinus empyema. Mendel also mentions cases of so-called retrobulbar neuritis which healed spontaneously or were cured by simple treatments. In the case of Coppez,[167] the bulb was turned outward with slight exophthalmus, derangement of vision, left opticus indistinct, suppuration of sphenoid sinus and sequestrum 1 cm. long. Mendel describes a case of unilateral retrobulbar neuritis which, after the suppurating ethmoid cells were opened, showed distinct improvement. In the case of Bergmeister-Hajek,[168] one-sided hyperemic neuritis, empyema of the ethmoid labyrinth resulted in cure. In the case of Fliess[169] retrobulbar neuritis, sphenoid sinus empyema of the right side, treatment was crowned by success. In the case of Lor[170] sphenoid sinus empyema and neuritis, with sudden blindness, operation, cure. Nuel[171] observed three similar cases of neuritis or neuroretinitis. In the case of Hoffman[172] treatment of empyema of the sphenoid sinus and the ethmoid cells was successful. In the case of Holmes[173] loss of the eyesight of the left eye, empyema of the left sphenoid sinus, opening, cure effected. In the case of Pauncz[174] the papillitis nervi optici showed decided improvement after the ethmoid cells were opened and washed out. In the case of Schmiegelow[175] the eyesight showed decided improvement after an operation for empyema of the sphenoid sinus and the last ethmoid cells. In the case of Fuchs-Hajek[176] cure of a retrobulbar neuritis with central scotoma was effected after the removal of the polypoid degenerated mucosa of the left posterior ethmoid cells and of the sphenoid sinus.

Concerning sight derangements of both sides and the diseased accessory cavities of both sides, we have but few cases at our command. Mendel mentions a case of double-sided optic nerve disease, caused by empyema of the ethmoid cells, giving no further detail than his source (English literature) on the subject. We saw a case of multiple accessory cavity suppuration and double atrophy of the optic nerves. In the case of Pollatschek[177] blindness on both sides from empyema of the sphenoid sinuses, was assumed. In the discussion we doubted the causal connection.[178] In the case of F. R. Alexander[179] suppuration of the left sphenoid sinus and the right ethmoid cells, papillitis on both sides; after opening the sinuses, a cure was effected. In the case of Schmiegelow[180] double empyema of the sphenoid sinus and the ethmoid cells, double neuritis, an operation, and marked improvement of sight. In the case of Delneuville[181] sight derangements on both sides, pus in the middle meatus. There was probably diseased sphenoid sinuses with or without conjunction of the ethmoid cells. After a month of purely medicinal treatment a cure was effected. The described intimate relation of the canalis opticus, sulcus opticus, the optic nerve and the chiasm to the posterior ethmoid cell, to the sphenoid sinus on one side, and also on both sides, the varying course of the canalis opticus with the optic nerve in the sinuses, the ethmoidal veins often lying free in the semicanalis ethmoidalis, the walls, thin as tissue paper, of the bone partitions, all these give an anatomic foundation for the explanation of the given observations.

For the origin and explanation of contralateral and double-sided sight derangements and blindness, with one-sided diseases of the accessory cavities, the etiologic causes already mentioned and the anatomic foundation mentioned by us may be useful. Our observations bear on the following cases:

1. The left posterior ethmoid cell constitutes the medial wall of the right canalis opticus.
2. The right posterior ethmoid cell constitutes the inferior and medial wall of the canalis opticus on both sides, and the wall of the entire sulcus opticus.

3. The right posterior ethmoid cell constitutes the wall of the entire sulcus opticus.

4. The right posterior ethmoid cell constitutes the wall of the right third and the middle third of the sulcus opticus.

5. The left sphenoid sinus constitutes the inferior wall of the right canalis opticus.

6. The right sphenoid sinus borders medially only the left optic nerve.

7. The left sphenoid sinus constitutes the inferior wall of the right canalis opticus, and the wall of the right third and the middle third of the sulcus opticus.

8. The left sphenoid sinus constitutes the inferior and medial wall of the canalis opticus on both sides, and the wall of the entire sulcus opticus.

9. The same morphologic relation with this difference, that only a small part of the right inferior and medial wall of the canalis opticus is constituted by the left sphenoid sinus; the larger part is constituted by the right posterior ethmoid cell.

10. The left sphenoid sinus constitutes the inferior and medial wall of the left canalis opticus, and the inferior wall of the right canalis opticus, also the wall of the entire sulcus opticus.

11. The right sphenoid sinus constitutes the wall of the middle third of the sulcus opticus.

The clinical material known to us has not been abundant. In the case of Wohlmuth[182] the right frontal and the orbital edge show signs of injury, bruise of the back of the head blindness of left eye confirmed. Berlin[183] assumed that the fracture of the right orbital roof extended along the left side, in this way striking the left canalis opticus or the indirect fracture was due to the injury of the occiput.

In the case of Freudenthal[188] after a Killian operation on the right side of the frontal, the left eye became blind and six weeks afterwards atrophy of the optic nerves was evident. The ophthalmologists, Oppenheimer and May, considered that in consequence of the Killian operation, there was an indirect fracture in the region of the left canalis opticus. Freudenthal, having no ground for other causes, also assumed this to be the case. In the case of Laas and Lewy, Lewy removed a piece of nasal septum on the left side 4 cm. long, 2½ cm. wide, which lay obliquely from below backwards. A quarter of an hour after the operation there were scintillations before the right eye and lessened sight. Upon examination Laas found almost total defect of the superior field of vision, normal fundus, the right papilla beginning to become pale; in two weeks the right opticus was clear white. Laas and Lewy were of the opinion that there was a fracture of the right canalis opticus. By correspondence I explained this case on the assumption that there was a fracture of the right canalis opticus. Pointing to this is the sudden appearance of sight impairment; my anatomic findings, which show the free course of the canalis opticus in the sphenoid sinus and the rear ethmoid cell, also those preparations which in the Atlas figures 14, 15, 67, 72, 73 and 91 illustrate, and which show the spreading out of the sphenoid sinuses between the lamellae of the nasal septum; furthermore, the spreading out of the sphenoid sinus, and of the posterior ethmoid cell from one side over the partition wall to the other side. These findings may explain an indirect fracture of the canalis opticus in the given case.[185] As regards the cases of Wohlmuth, Freudenthal, and Laas and Lewy, it is impossible to deny the probable existence of an indirect contralateral fracture, in the region of the canalis opticus, the foramen opticum, and the sulcus opticus, considering the fact that indirect fractures of the canalis opticus on the same side as the injuries have often been observed, and cases under our notice have shown that the posterior ethmoid cell as well as the sphenoid sinus may be separated from the opposite canalis opticus by a bony wall of paper-like thinness. Hölder has observed fifty-three cases of fracture of the walls of the canalis opticus. Should an indirect fracture arise, in view of the existing relations observed by us, its position must be contralateral. But if we give our closer attention to the Freudenthal case, two facts must give food for reflection: first, in spite of numerous operations on the skull, done with hammer and chisel, the history of not a single case is known; second, the patient's frontal was first opened according to Kuhnt's method, and the assumed indirect contralateral fracture must have, in consequence, appeared after the subsequent demolition of the inferior frontal wall. In all operations performed according to the Killian method, in which the use of hammer and chisel is more general, than in this case, traumatic lesion of the optic nerve on the same side or on the contralateral has not been observed. We could not forego these few remarks, although in consequence of our described investigations, we declared an indirect contralateral fracture of the canalis opticus possible. In the case of Halstead[186] there were empyema of the maxillary sinus, the ethmoid cells and sphenoid sinus of the right side with blindness of the left eye. Halstead thought that there was a rupture of the empyema of the right sphenoid sinus into the left sphenoid sinus, in consequence of which an old left-sided neuritis responded. Brown thought that there was exudation in the left optic sheath; half a year after the operation the left eye was normal. Rhinoscopically the rupture of the empyema of the right sphenoid sinus into the left one was not proven, and the ophthalmoscopic findings could not establish the presence of an old neuritis. We must mention the existence of an empyema of the ethmoid bone, and in consequence it may be assumed that there is a causal connection as well with the empyema of the ethmoid cells. According to our morphologic findings, transmission of the inflammation from the posterior ethmoid cell, as well as the sphenoid sinus to the contralateral optic nerve is possible, as well as a circulatory disturbance; without hypotheses this is given as the reason for contralateral sight impairments. In the case of Glegg and Hay[187] with empyema of the right ethmoid cells there occurred hemianopsia bitemporalis, and paralysis of the associated movements. Cure was due to operative measures. They took the empyema to be the direct cause of the lesion of the chiasm and the crossing of the fibres of the Gudden commissure caused the associated derangements. Our morphologic findings make lesion of the chiasm possible from diseases of the posterior ethmoid cells as well as the sphenoid sinuses. The part which the Gudden commissure plays in the associated movements comes within the province of the neurologist and ophthamologist. In the case of Pollatscheck,[188] with empyema of the left posterior ethmoid cells and sphenoid sinus, papillitis on both sides, a cure was effected by operation. He thought that there was bony dehiscence of the sphenoid sinus on only one or both sides, and that the resultant periostitis extended per continuitatem to the optic nerve on the other side Concerning this case, in his description he says that upon opening the posterior ethmoid cells a great deal of fetid pus was evacuated, but on opening the sphenoid little pus came out; the explanation rests on the hypothesis of a sphenoid sinus having bony dehiscences on one or both sides, and of a periostitis, with transmission to the optic nerve. Data are completely lacking concerning the extent of the opened posterior ethmoid cells, and the sphenoid sinus, and the reasons for exenterating the posterior ethmoid cells. Our morphologic observations admit the possibility of a causal connection with the diseased posterior ethmoid cells as well as diseases of the sphenoid sinus; still the question arises, as to the accidental spontaneous appearance of acute one-sided empyema and papillitis of both sides. In the case of Polyak[189] a bone cyst on the left side and latent multiple accessory cavity empyemas were accompanied by exophthalmus and atrophia optica on both sides; a year earlier there was disease of the right eye. He considered that the pus from the bone cyst forced its way successively into the left accessory cavities, then into those of the right side, and finding no way of flowing off, caused the dilation of the accessory cavities and pressure. In this way he explains the exophthalmus and atrophy of the optic nerves on both sides. We believe that Polyak's explanation does not hold good. The observations made by Goldzieher and myself, which had for their object a more plausible interpretation, we shall leave unmentioned, as we have recently learned that a necroscopic investigation is under way, the result of which is finally to settle this point. In the case of Gutmann,[190] empyema of the left sphenoid sinus was associated with blindness on the right side. He believes that a transmission to the ethmoid cells and the sphenoid sinus caused an amaurosis.

In our case, besides left-sided empyema of the posterior ethmoid cells, and the sphenoid sinus, earlier atrophia optica of the right side, neuritis optica with hemianopsia temporalis of the left side, also existed. After an operation, the eye showed a slight improvement, vision 6/15 to 6/10. Six years before a large number of polypi had been taken out of the left nasal cavity, then there was a suppurative fetid discharge on the left side, with constant headaches; in the course of two months the right eye became blind; three months later the sight of the left eye was impaired, and investigation showed a temporal hemianopsia. Slight improvement only was reported after operation. Lues was not indicated, as antiluetic treatment proved negative. We illustrate in figure 33 the temporal hemianopsia of the left eye. The patient did not remain longer under observation, so I cannot report further as to the fate of the left eye. Prof. Szily left the case to me, and we decided that the causal connection was entirely possible; there seemed no reason for any other construction, besides, it corresponds to our morphologic findings. I possess a preparation, in which the left sphenoid sinus only constitutes the wall of the right canalis opticus, and the wall of the middle third of the sulcus opticus. A right-sided atrophy, and left-sided temporal hemianopsia would be explained anatomically by a lesion of the right optic nerve and of the chiasm by a diseased left sphenoid sinus. The accidental appearance of both diseases spontaneously can only be explained by necroscopic investigation.

Concerning contralateral sight impairment, we have at our command no postmortem findings. The morphologic relations we have observed constitute the anatomic foundation explaining the lesion of the contralateral optic nerve and the chiasm, furthermore, that of the chiasm or both optic nerves in a disease of one side of a posterior ethmoid cell, or of a sphenoid sinus.

In regard to the therapy of diseases of the individual cavities, I refer to our work,[101] in which I have described in detail on pages 236 to 401 the treatment of individual cavities, and the different courses of procedure. In addition, on pages 382 to 401, I have devoted special chapters to the indications for interference in the treatment of the complications of the individual accessory cavities.

I wish to make a supplementary statement. I have, in general, called attention to the latent foci, and their explorations, but must emphasize that fact that in those cases of sight derangement, in which the oculist cannot confirm the cause, and the suspicion of nasal connection exists, even though the findings be negative, it seems well to take explorative measures, and to seek the cause, whether or not the presence of latent foci is suspected. The good success of Schmiegelow and others strengthen us in our belief. I have never failed to use this test in cases given over to our care, and if I was not always successful in locating latent foci, at least I felt nothing had been neglected. I herewith close my clinical observations.

The causal connection of a one-sided, double-sided or contralateral sight impairment and blindness in diseases of the accessory cavities, particularly those of posterior ethmoid cells and the sphenoid sinus, is conclusively proven; the anatomic foundation of this theory is sufficiently established by reason of my morphologic findings; they allow of every natural conclusion, without making use of any forced or improbable hypotheses. An exact pathologico-anatomical foundation is still to be created.

I am convinced that a complete elaboration of this theory would be made possible by exact clinical observations, and through necroscopic investigations, that mutual rhinologic and ophthalmologic researches will close this important and interesting chapter of medicine.

REFERENCES.

1. Berger and Tyrmann. Die Kranheiten der Keilbeinhöhle und des Siebbeinlabyrinthes, 1886
2. Zuckerkandl. Anatomie der Nasenhöhle, 1882, 1893.
3. Douglas. Monatsschrift für Ohren- und Kehlkopfkranheiten, 1897.
4. Onodi. Die Nebenhöhlen der Nase, 1905.
5. Onodi. Die Dehiszenzen der Nebenhöhlen der Nase. Archiv. für Laryngologie, Bd. xv.
6. Zuckerkandl. Anatomie der Nasenhöhle, 1893.
7. Merlin. Berichte des Naturwissenschaftlich-medizinischen Vereines in Innsbruck, 1884-1885, 1885-1886.
8. Zuckerkandl and Helly. Deutsche Zeitschrift für Chirurgie, Bd. iii.
9. Denouvilliers and Gosselin. Compend. de chir. prat., 1852.
10. Winkler. Archiv. für Laryngolgie, 1894.
11. Killian. Münchener medizinische Wochenschrift, 1897.
12. Hyrtl. Vergangenheit und Gegenwart des Museums für menschliche Anatomie, 1869.
13. Spee. Bardelebens Handlbuch der Anatomie, 1896.
14. Gallmaerts. Annal. d. Ocul., 1900.
15. Holmes. Arch. of Ophthalm., 1896.
16. Onodi. Archiv. für Laryngologie, Bd. xiv und xv.
17. Onodi. Archiv. für Laryngologie, Bd. xi.
18. Onodi. Archiv. für Laryngologie, Bd. xv.
19. Bering and Wicherkiewicz. Berliner klinische Wochenschrift, 1882.
20. Schmidt-Rimpler. Archiv. für Laryngologie, Bd. xvii.
21. Onodi. Archiv. für Laryngologie, Bd. xvii.
22. Virchow. Krankhafte Geschwülste.
23. Manz. Heidelberger Berichte, 1887.

24. Enslin. Graefes Archiv für Ophthalmologie, Bd. lviii.
25. Goldzieher. Budapesti orvosi ujság, Szemeszet, 1904.
26. Hölder. Berlin, Graefe-Sämisch Handbuch.
27. Ortmann. Virchow's Archiv, Bd. cxx.
28. Hajek. Archiv. für Laryngologie, Bd. xviii.
29. Westenhöfer. Berliner klinische Wochenschrift, 1905.
30. Westenhöfer. Deutsche medizinische Wochenschrift, 1906.
31. Foucher. Courtaix, Recherches cliniques sur les relations patholog. entre l'oeil et les dents, Paris, 1895.
32. Mair. Edinburgh Med. Journ., 1866.
33. Panas. Archiv. d' Ophthalmol., 1885.
34. Dmochovszky. Archiv. für Laryngologie, 1895, Bd. iii.
35. Westermayer. Münchener medizinische Wochenschrift, 1895.
36. Bousquet. Progrés Medical, 1877.
37. Koehler. Charite-Annalen, 1892.
38. Schindler. Archives de méd. et de pharm.-militaires, 1892.
39. Zirm. Wiener medizinische Wochenschrift, 1892.
40. Huguénin. Korrespondenzblatt der Schweizer Aerzte, 1882.
41. Müller. Wiener klinische Wochenschrift, 1895.
42. Paulsen. Hospitals Tidende, 1861.
43. Knapp. Archiv. für Augenheilkunde, 1880.
44. Bourat-Lécard. Bordeaux méd., 1875.
45. Redtenbacher. Wiener medizinische Blätter, 1892.
46. Lennox-Browne. Journal of Laryngol., Vol. vii.
47. Hoppe. Klinische Monatsblätter für Augenheilkunde, 1893.
48. Krecke. Münchener medizinische Wochenschrift, 1894.
49. Wallenberg. Neurologisches Zentralblatt, 1895.
50. Silex. Berliner klinische Wochenschrift, 1896.
51. Milligan. The Medical Chronicle, 1899.
52. Fränkel. Virchow's Archiv, Bd. cxliii.
53. Macewen-Millar-Killian. Verein süddeutscher Laryngologen, 1900.
54. Roth. Wiener klinische Wochenschrift, 1899.
55. Carver. British Med. Journ., 1883.
56. Cholle. Archives internat. de laryng., 1906.
57. Begbie. Med. Times and Gaz., 1852.
58. Schäffer. Prager medizinisches Wochenschrift, 1883.
59. Pauncz. Archiv. für Laryngologie, Bd. xiii.
60. Jacubasch. Berliner klinische Wochenschrift, 1875.
61. Ogston. British Med. Journ., 1885.
62. Warner. British Med. Journ., 1885.
63. Ewald. Deutsche medizinische Wochenschrift, 1890.
64. Trousseau. Clinique médical. Deutsche, 2. Aufl., 1866.
65. Hajek. Archiv. für Laryngologie, Bd. xviii.
66. Horner. Klinische Monatsblätter für Augenheilkunde, 1863.
67. Panas. Soc. de Chir. de Paris, 1873.
68. Rouge. L' Union médic., 1872.
69. Raymond. Bull. Soc. Anatom., Paris, 1895.
70. Grünwald. Die Lehre von den Naseneiterungen, 1893.
71. Vince. Tod. La Clinique, 1900.
72. Thiroloix. Bull. Soc. Anat., 1892.
73. Flatau. Nasenkrankheiten, etc., 1895.
74. Zöckendörfer. Prager medizinische Wochenschrift, 1893.
75. Pekastovsky. Internationales Zentralblatt. für Laryngologie. Bd. x.
76. Scholz. Berliner klinische Wochenschrift, 1892.

77. Schlagenhaufer. Wiener klinische Wochenschrift, 1899.
78. Pauncz. Zeitschrift für Augenheilkunde, 1906.
79. Halász. Orvosi Hetilap, 1904.
80. Schroeder. Zeitschrift für Ohrenheilkunde, 1906.
81. Weichselbaum. Wiener medizinische Wochenschrift, 1890.
82. Pauncz. Orvosi Hetilap.
83. Hansberg. Zeitschrift für Ohrenheilkunde, Bd. xliv.
84. Duplay. Arch. gén. de méd., 1874.
85. Russel. Med. Times and Gaz., 1878.
86. Vossius. Zeitschrift für Augenheilkunde, 1900.
87. Holmes. Archives of Ophthalmology, 1886.
88. Demarquay. M. Mackenzie, Die Krankeiten der Nase, etc., 1884.
89. Panse. Archiv. für Laryngologie, l. c.
90. Leber. Archiv. für Laryngologie, Bd. xvii.
91. Finlag. Monatsschrift für Ohrenheilkunde, 1905.
92. Kuhnt. Entzündliche Erkrankungen der Stirnhöhle, 1895.
93. Kuhnt und Gurwitsch. Graefes Archiv, Bd. xxix.
94. Krauss. Münchener medizinische Wochenschrift, 1907.
95. Nolthenius. Monatsschrift für Ohrenheilkunde, 1895.
96. Treitel. Verhandlungen der Laryngologischen Gesellschaft, Berlin, 1900.
97. Caldwell. Med. Journ. of New York, 1898.
98. Grossmann. Weinlechner, Bereicht. der K. K. Krankenanstalt Rudolfstiftung, 1875.
99. Depaques. Bull. de la soc. d' ophth. de Paris, 1895.
100. Körner. Verhandlungen der Deutschen odontologischen Gesellschaft, Bd. vii.
101. Le Fort. France méd., 1876.
102. Pagenstecher. Archiv. für Augenheilkunde, 1884.
103. Brück. Wochenschrift für die gesamte Heilkunde, Berlin, 1851.
104. Pasquier. Lanc. franc., 1839.
105. Buzer. Berliner klinische Wochenschrift, 1868.
106. Galezowski. Archives inter. de Laryng., 1905.
107. Onodi. Revue hebdomadaire de laryngol., etc., 1895.
108. Grünwald. Lehre von der Naseneiterung, 2. Aufl.
109. Panzer. Wiener klinische Wochenschrift, 1896.
110. Tedenat. Soleville, Thése de Montpelier, 1890.
111. Westermayer. Münchener medizinische Wochenschrift, 1895.
112. Bournonville. Zentralblatt für Chirurgie, 1885.
113. Jansen. Archiv. für Laryngologie, Bd. i.
114. Flatau. Rev. int. de rhin., 1893.
115. Lichtwitz. Annal. de malad. d'or., 1896.
116. Killian. Heymann's Handbuch, Bd. iii.
117. Dmochowski. Archiv. für Laryngologie, Bd. iii.
118. Bauby. Arch. d. ophthalm., 1897.
119. Salva. Thése de Paris, 1895.
120. Schmiegelow. Archiv. für Laryngologie, Bd. xvi.
121. Jacqueau. Internationales Zentralblatt für Laryngologie, Bd. xvi.
122. Lapersonne. Bull. de la société franc. d'opth., 1902.
123. Spencer-Watson. Diseases of the Nose and Its Accessory Cavities.
124. Panas. Progrès méd., 1887.
125. Baümler. Kongress für innere medizin, 1890.

126. Steinthal. Medizinisches Korrespondenzblatt der Württembergischen arztlichen Landesvereine, 1891.
127. Köhler. Charité Annal., 1891.
128. Botey. Revue de laryng., 1897.
129. Burger. Zentralblatt für Laryngologie, Bd. xi.
130. Caldwell. Zentralblatt für Laryngologie, Bd. x.
131. Hajek. Erkrankungen der Nasenhöhlen der Nase, 1903.
132. Hjorth. Schmiegelow, Archiv. für Laryngologie, Bd. xv.
133. Onodi. Archiv. für Laryngologie, Bd. xvii.
134. Baurowitz. Archiv. für Laryngologie, Bd. xii.
135. Mann. Verhandlungen der Deutschen otologischen Gesellschaft, 1901.
136. Schmiegelow. Archiv. für Laryngologie, Bd. xv.
137. Eversbusch. Graefe-Sämisch Handbuch, 2 Aufl.
138. Baumgarten. Orvosi Hetilap, 1903.
139. Post. Lancet, 1882.
140. Sakos und Luc. Revue de laryngol., 1899.
141. Broeckart. Revue de laryngol., 1901.
142. Hoffmann. Zeitschrift für Augenheilkunde, 1906.
143. Alexander. Verhandlungen der Deutschen otologischen Gesellschaft, 1905.
144. Baratoux. Arch. ital. di laryng., 1883.
145. Flatau. Zentralblatt für Laryngologie, 1894-1895.
146. Foucher. Zentralblatt für Laryngologie, 1894-1895.
147. Schäffer. Deutsche medizinische Wochenschrift, 1892.
148. Onodi. Archiv. für Laryngologie, l. c.
149. Hinkel. American Laryng. Assoc., 1902.
150. Ziem. Berliner klinische Wochenschrift, 1888.
151. Berger. Rapports entre les malad. des yeux, etc., 1892.
152. Bryan. American Laryng. Assoc., 1895.
153. Häffner. Münchener medizinische Wochenschrift, 1904.
154. Onodi. Zeitschrift für Augenheilkunde l. c.; Archiv für Laryngologie, l. c.
155. Paunez. Archiv. für Augenheilkunde, Bd. lii.
156. Halstead. American Laryng., Rhin. and Otol. Soc., 1901.
157. Pollatschek. Zentralblatt für Laryngologie, Bd. xxii.
158. Mendel. Zentralblatt für Augenheilkunde.
159. Sattler. Onodi, Archiv. für Laryngologie, Bd. xvi.
160. Hirschberg. Zentralblatt für Augenheilkunde.
161. Grossmann. Allgemeine Wiener medizinische Zeitung, 1893.
162. Snellen. Med. Tidschr. vor Geneesk., 1894.
163. Knapp. Zeitschrift für Ohrenheilkunde, 1894.
164. Gronbäk. Verhandlungen des Dänischen oto.-laryngologischen Vereines, 1904.
165. Risley. Internationales Zentralblatt für Laryngologie, 1905.
166. Coppez und Lor. La Presse méd. Belge., 1900.
167. Coppez. Ophthalmologische klinik, 1902.
168. Hajek. Die Erkrankungen der Nebenhöhlen der Nase, 1903.
169. Fliess. Wiener klinische Rundschau, 1895.
170. Lor. Schmiegelow, Archiv. für Laryngologie, Bd. xv.
171. Nuel. Schmiegelow, Archiv. für Laryngologie, Bd. xv.
172. Hoffmann. Verhandlungen der Deutchen otologischen Gesellschaft, 1897.
173. Holmes. Archives of Ophthalmol., 1896.
174. Paunez. Zentralblatt für Laryngologie, Bd. xxii.
175. Schmiegelow. Archiv. für Laryngologie, Bd. xviii.

176. Fuchs-Hajek. Fuchs, Lehrbuch der Augenheilkunde, 1905.
177. Pollatschek. Ovorsi Hetilap.
178. Pottalschek. Ovorsi Hetilap.
179. Alexander. Verhandlungen der Deutschen otologischen Gesellschaft, 1905.
180. Schmiegelow. Archiv. für Laryngologie, Bd. xviii.
181. Delneuville. La Presse oto.-laryngologique Belge, 1906.
182. Wohlmuth. Berlin, Graefe-Sämisch, 1, Aufl.
183. Berlin. Idem.
184. Freudenthal. Archives internat. Chaveau et St. Hilaire, Paris, 1905.
185. Laas reported this case in the February meeting of the Berliner ophthalmologischen gesellschaft.
186. Halstead. Americ. Laryng., Rhin. and Otol Sol., 1901.
187. Glegg und Hay. Archiv. für Laryngologie, Bd. xvii.
188. Pollatschek. Zentralblatt für Laryngologie, Bd. xxii.
189. Polyák. Archiv. für Laryngologie, Bd. xv.
190. Gutmann. Zeitschrift für Augenheilkunde, 1906.
191 Onodi und Rosenberg. Die Behandlung der Krankheiten der Nase und des Nasen-Rachenraumes, 1906.

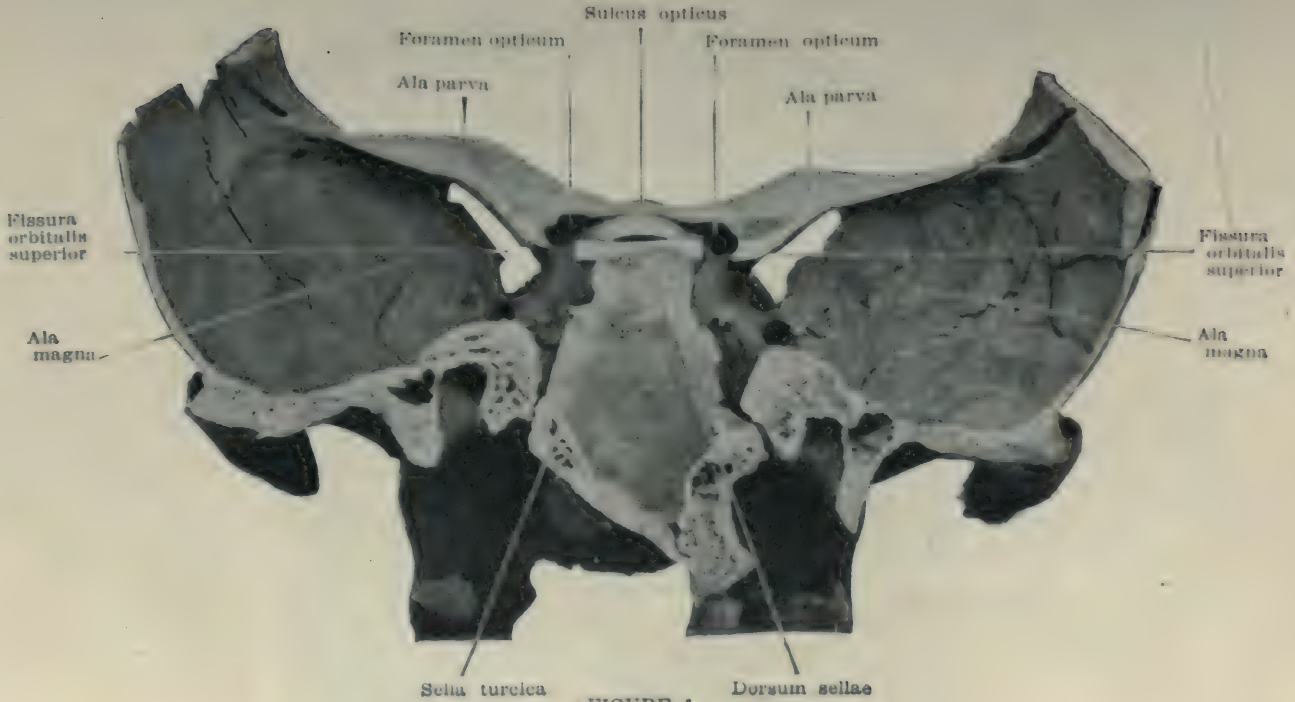
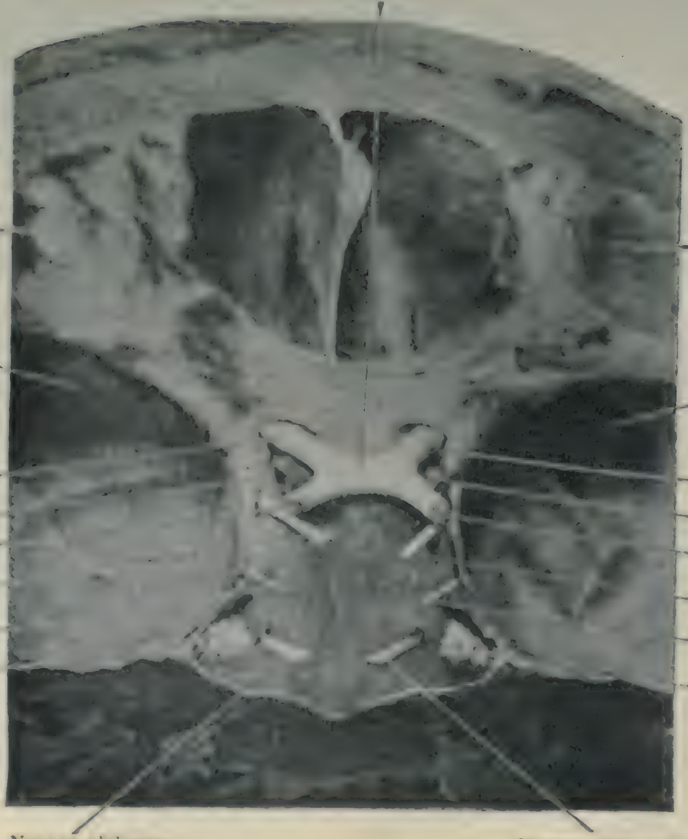
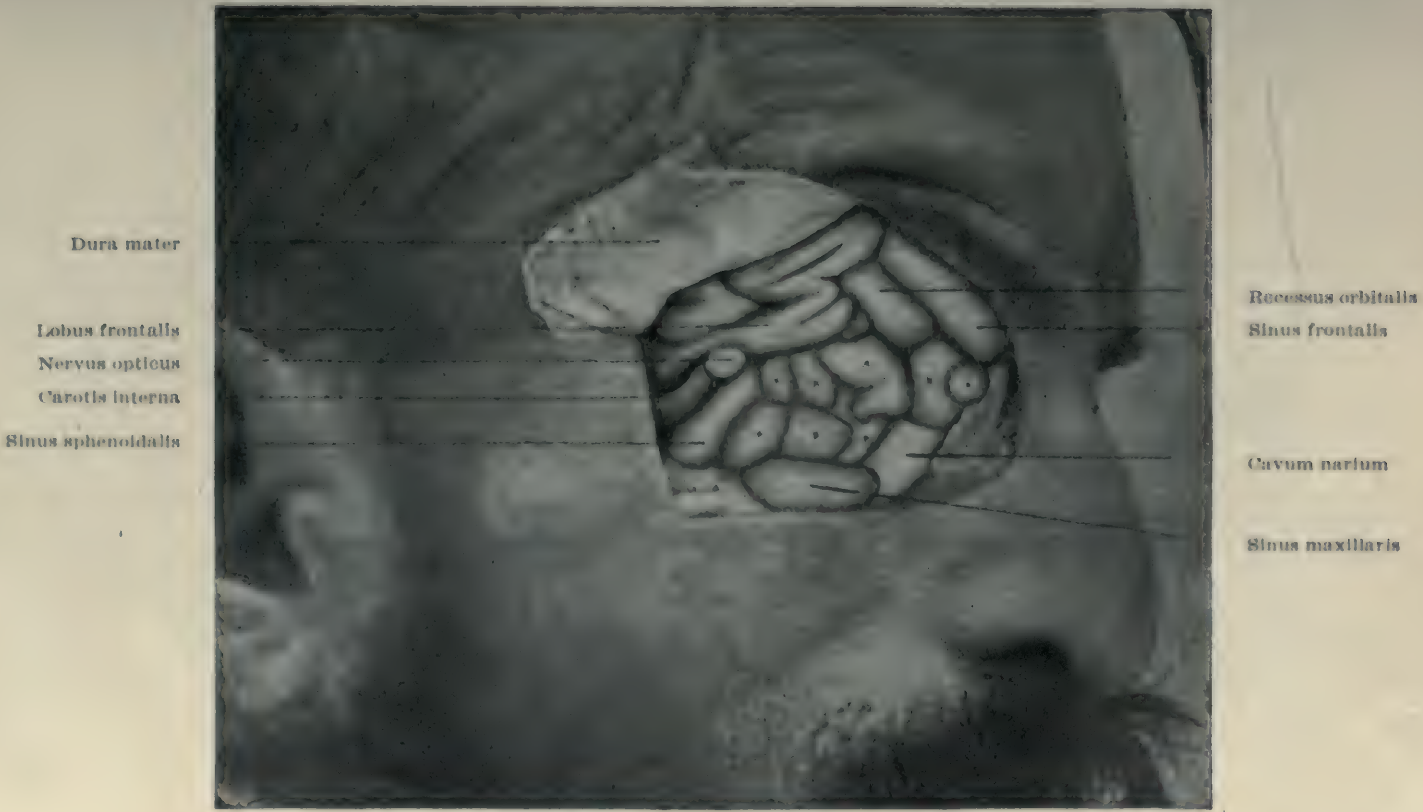

Sulcus opticus
Foramen opticum
Foramen opticum
Ala parva
Ala parva
Fissura orbitalis superior
Fissura orbitalis superior
Ala magna
Ala magna
Sella turcica
Dorsum sellae

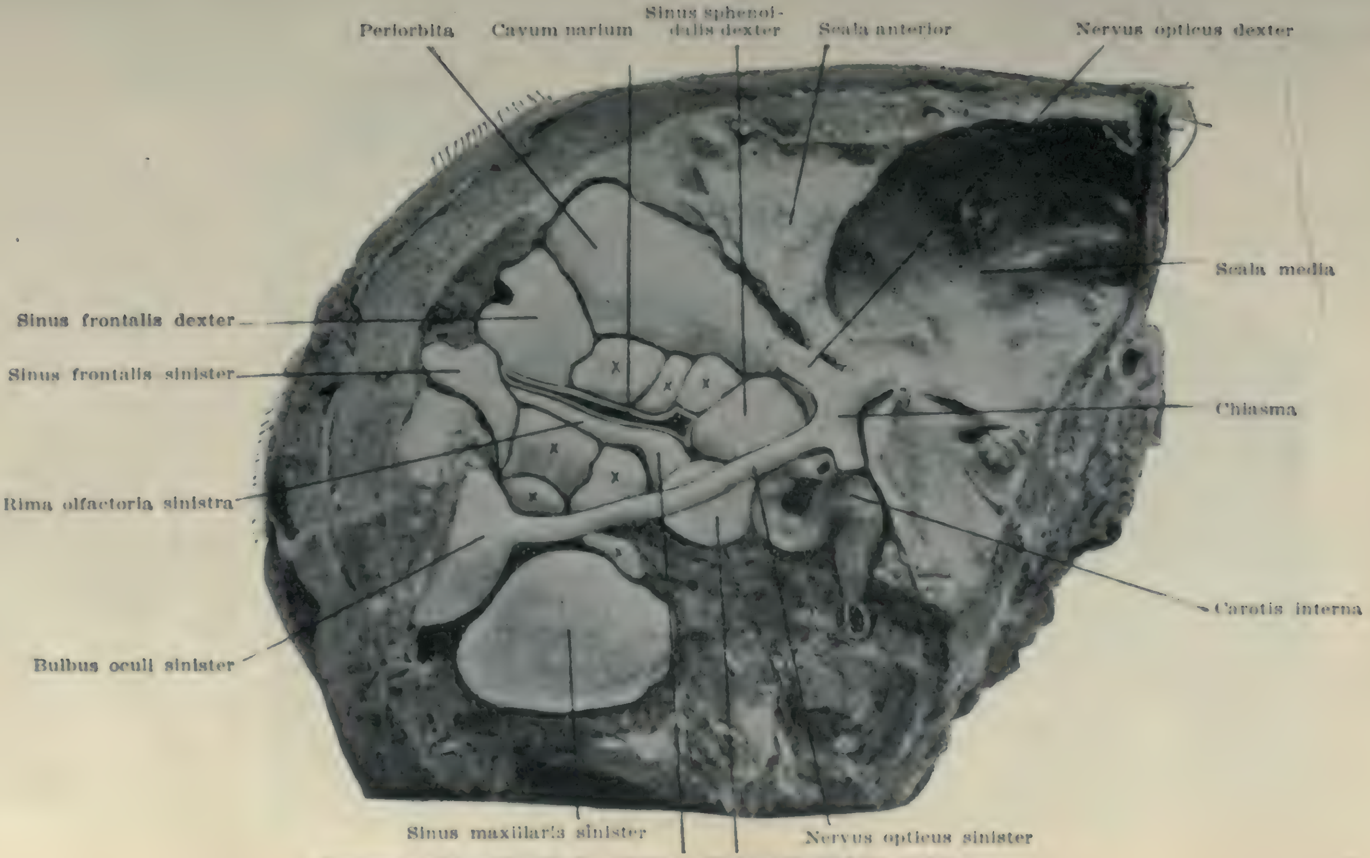
FIGURE 1.

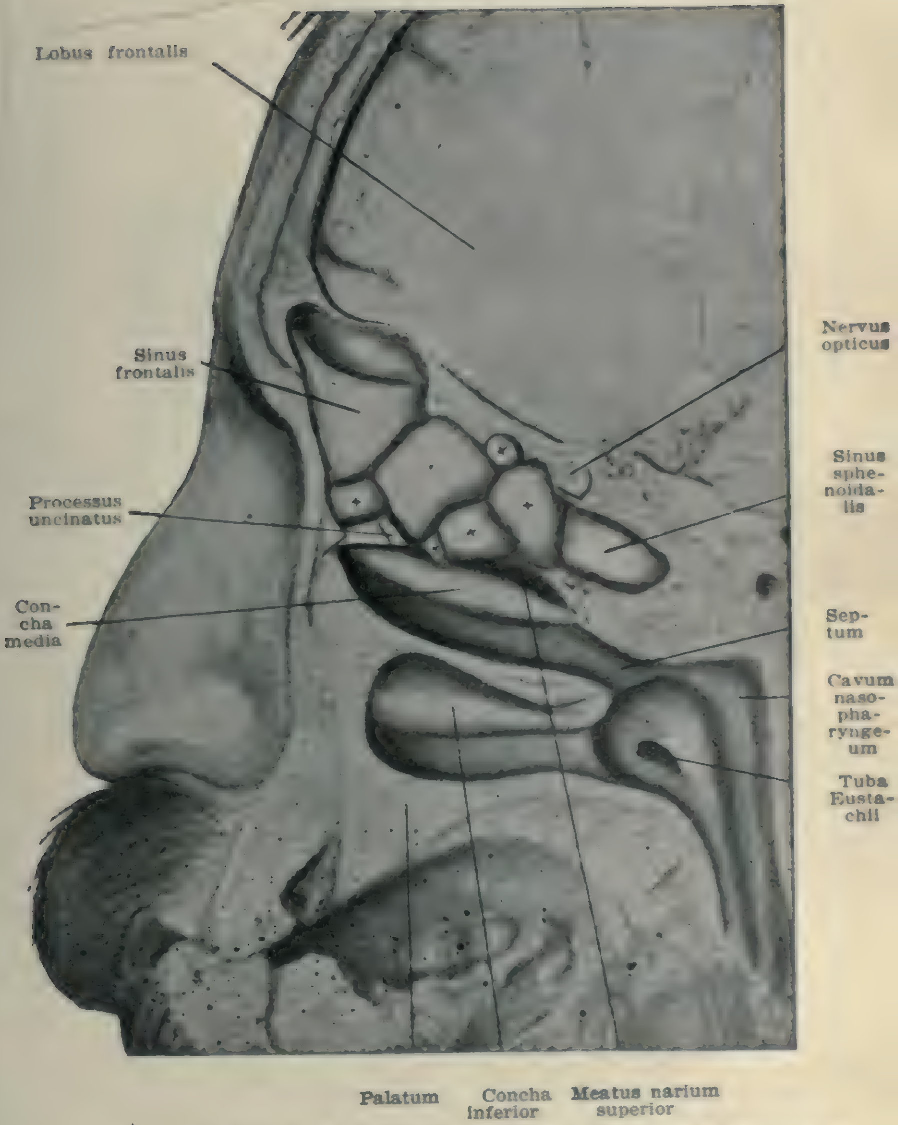

Chiasma
Scala anterior
Scala anterior
Scala media
Scala media
Nervus opticus sinister
Nervus opticus dexter
Carotis interna
Carotis interna
Tractus opticus sinister
Tractus opticus dexter
Nervus oculomotorius
Nervus oculomotorius
Nervus trochlearis
Nervus trochlearis
Nervus trigeminus
Nervus trigeminus
Nervus abducens
Nervus abducens



X X X X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURES 8 and 19


Periorbita
Cavum narium
Sinus sphenoidalis dexter
Scala anterior
Nervus opticus dexter
Scala media
Sinus frontalis dexter
Sinus frontalis sinister
Chiasma
Rima olfactoria sinistra
Carotis interna
Bulbus oculi sinister
Sinus maxillaris sinister
Nervus opticus sinister
Recessus sphenoethmoidalis sinister
Sinus sphenoidalis sinister
X X X X X X Cellulae ethmoidales anteriores et posteriores.



X X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 5.

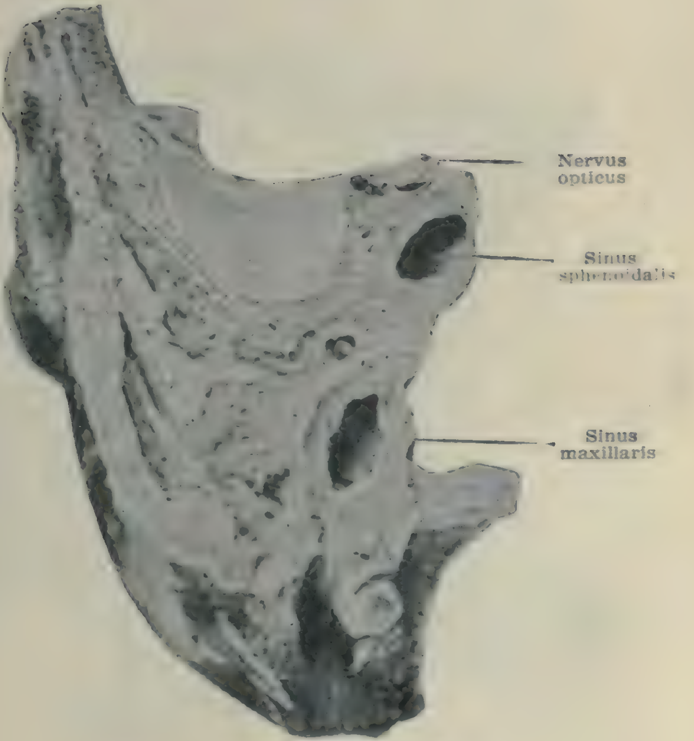


FIGURE 6.

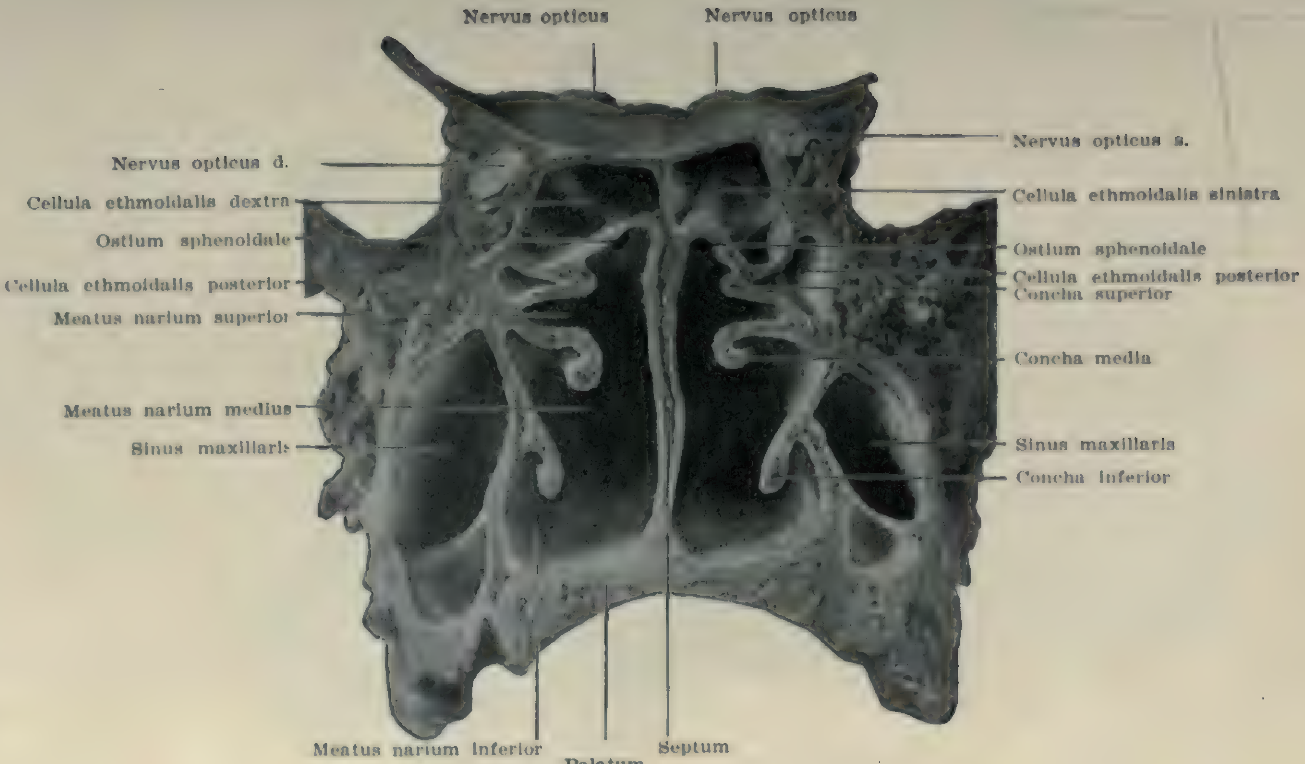

Nervus opticus
Nervus opticus
Nervus opticus d.
Nervus opticus s.
Cellula ethmoidalis dextra
Cellula ethmoidalis sinistra
Ostium sphenoidale
Ostium sphenoidale
Cellula ethmoidalis posterior
Cellula ethmoidalis posterior
Concha superior
Meatus narium superior
Concha media
Meatus narium medius
Sinus maxillaris
Sinus maxillaris
Concha inferior
Meatus narium inferior
Palatum
Septum

FIGURE 7.

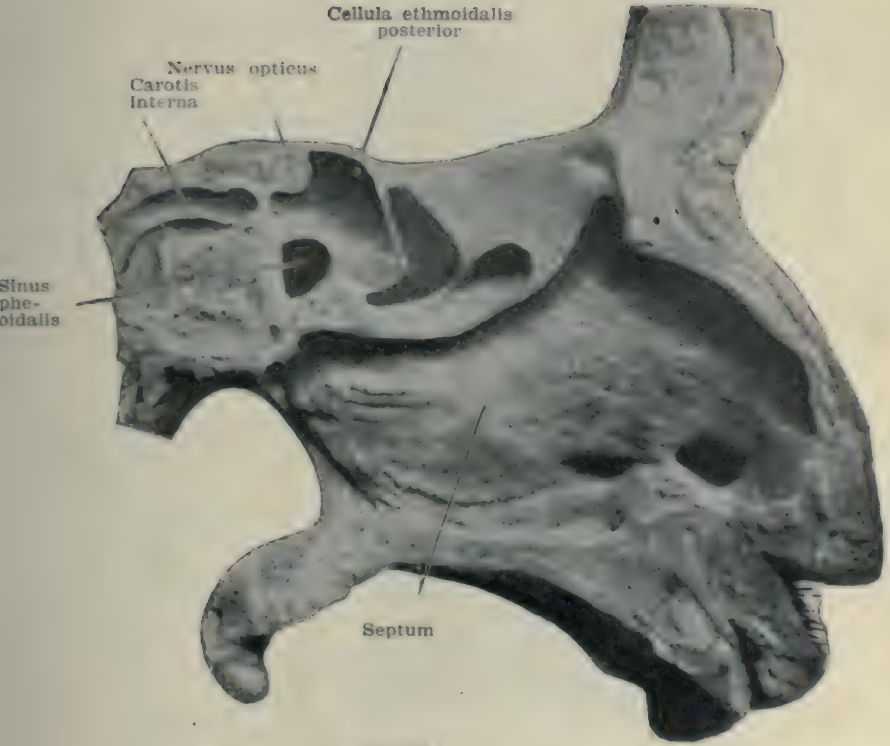

Cellula ethmoidalis posterior
Nervus opticus
Carotis interna
Sinus
phe-
oidalis
Septum

FIGURE 8.

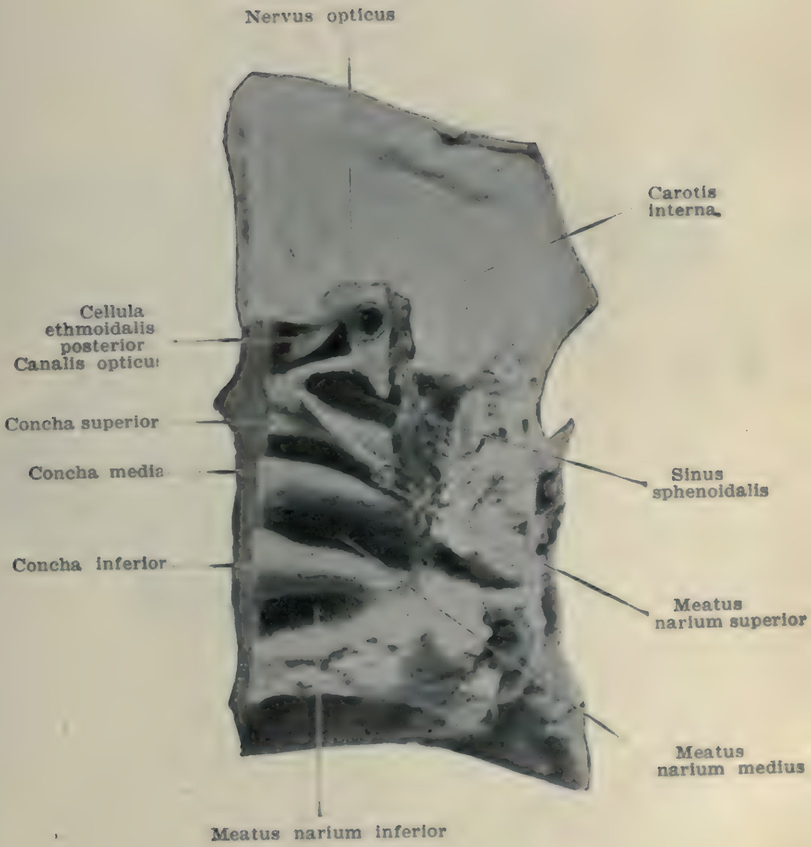

Nervus opticus
Carotis interna
Cellula ethmoidalis posterior
Canalis opticus
Concha superior
Concha media
Concha inferior
Sinus sphenoidalis
Meatus narium superior
Meatus narium medius
Meatus narium inferior

FIGURE 9.

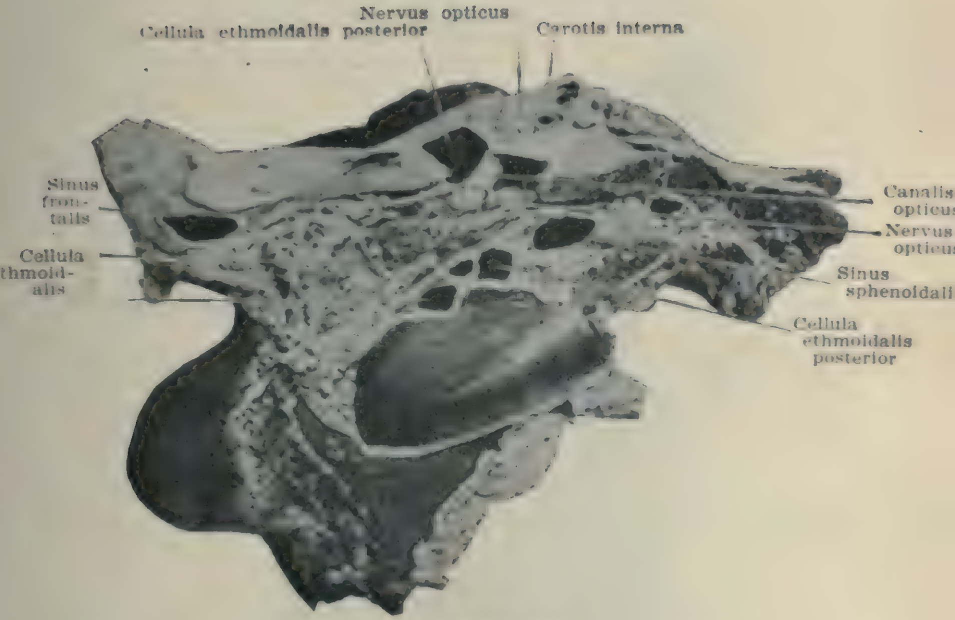

Nervus opticus
Cellula ethmoidalis posterior
Carotis interna
Sinus frontalis
Cellula thmoidalis
Canalis opticu
Nervus opticu
Sinus sphenoidali
Cellula ethmoidalis posterior

FIGURE 10.

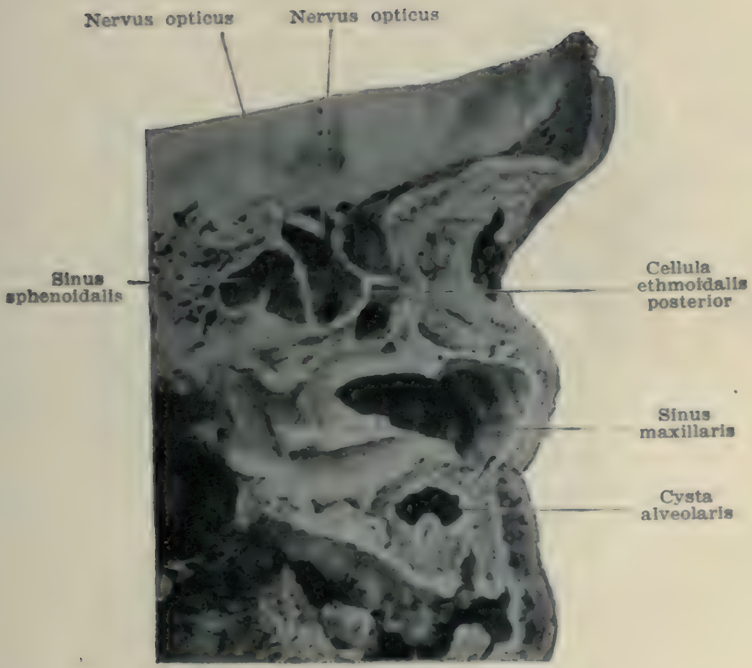

Nervus opticus
Nervus opticus
Sinus sphenoidalis
Cellula ethmoidalis posterior
Sinus maxillaris
Cysta alveolaris

FIGURE 11.

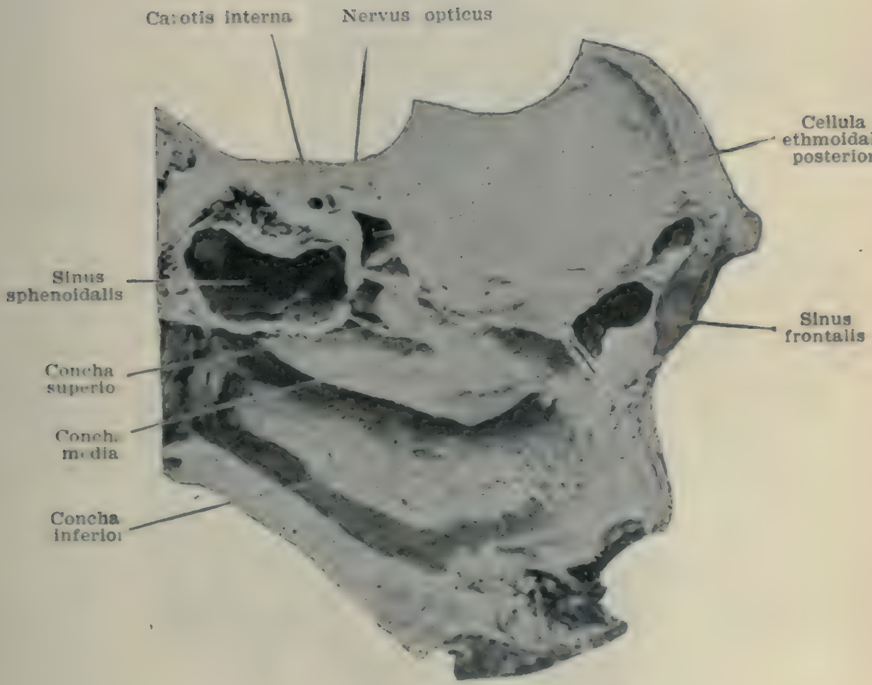

Carotis interna
Nervus opticus
Cellula ethmoidalis posterior
Sinus sphenoidalis
Sinus frontalis
Concha superior
Concha media
Concha inferior

FIGURE 12.

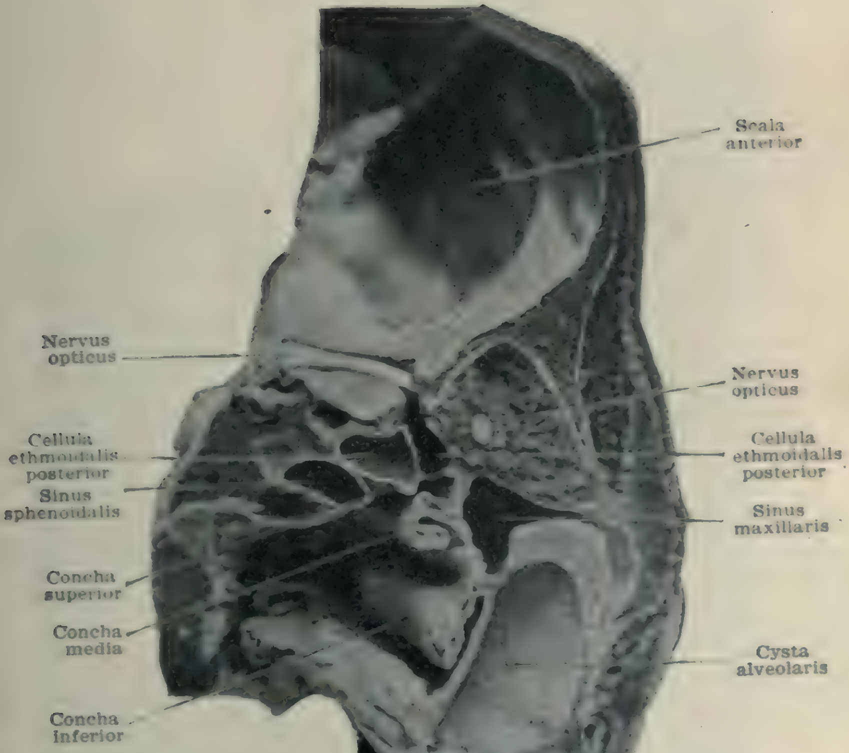

Scala anterior
Nervus opticus
Nervus opticus
Cellula ethmoidalis posterior
Cellula ethmoidalis posterior
Sinus sphenoidalis
Sinus maxillaris
Concha superior
Concha media
Cysta alveolaris
Concha inferior

FIGURE 13.

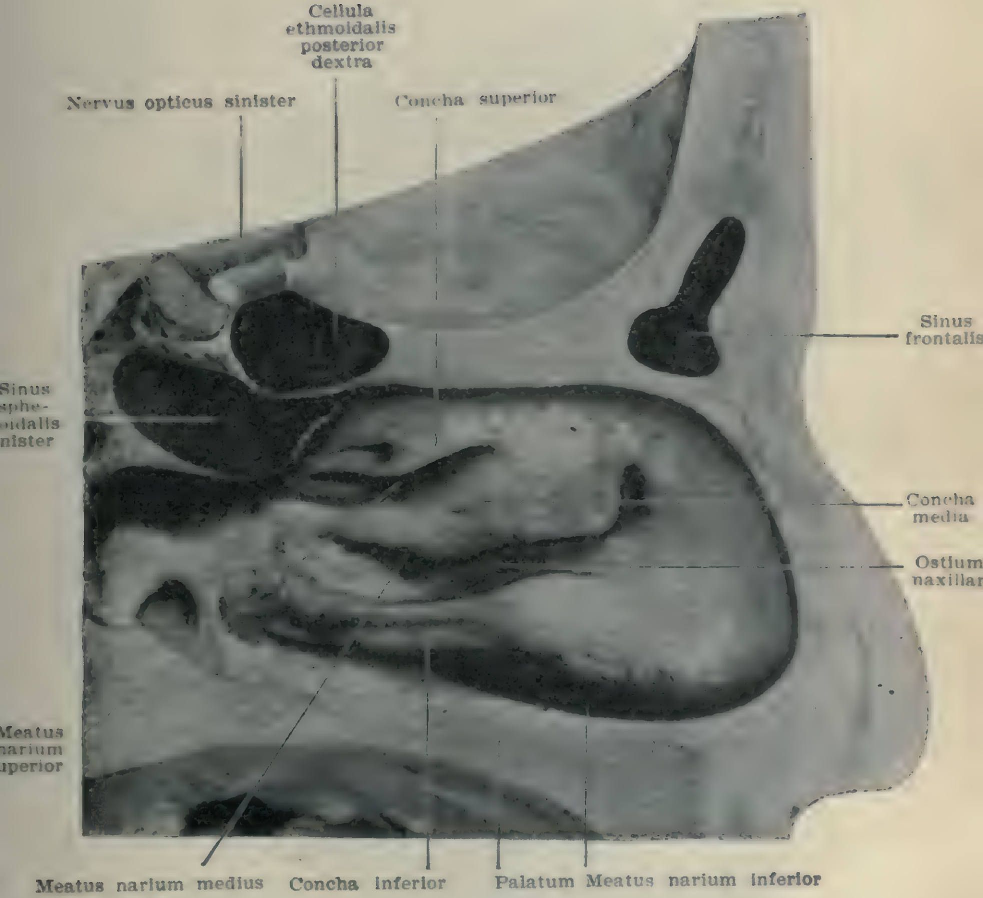
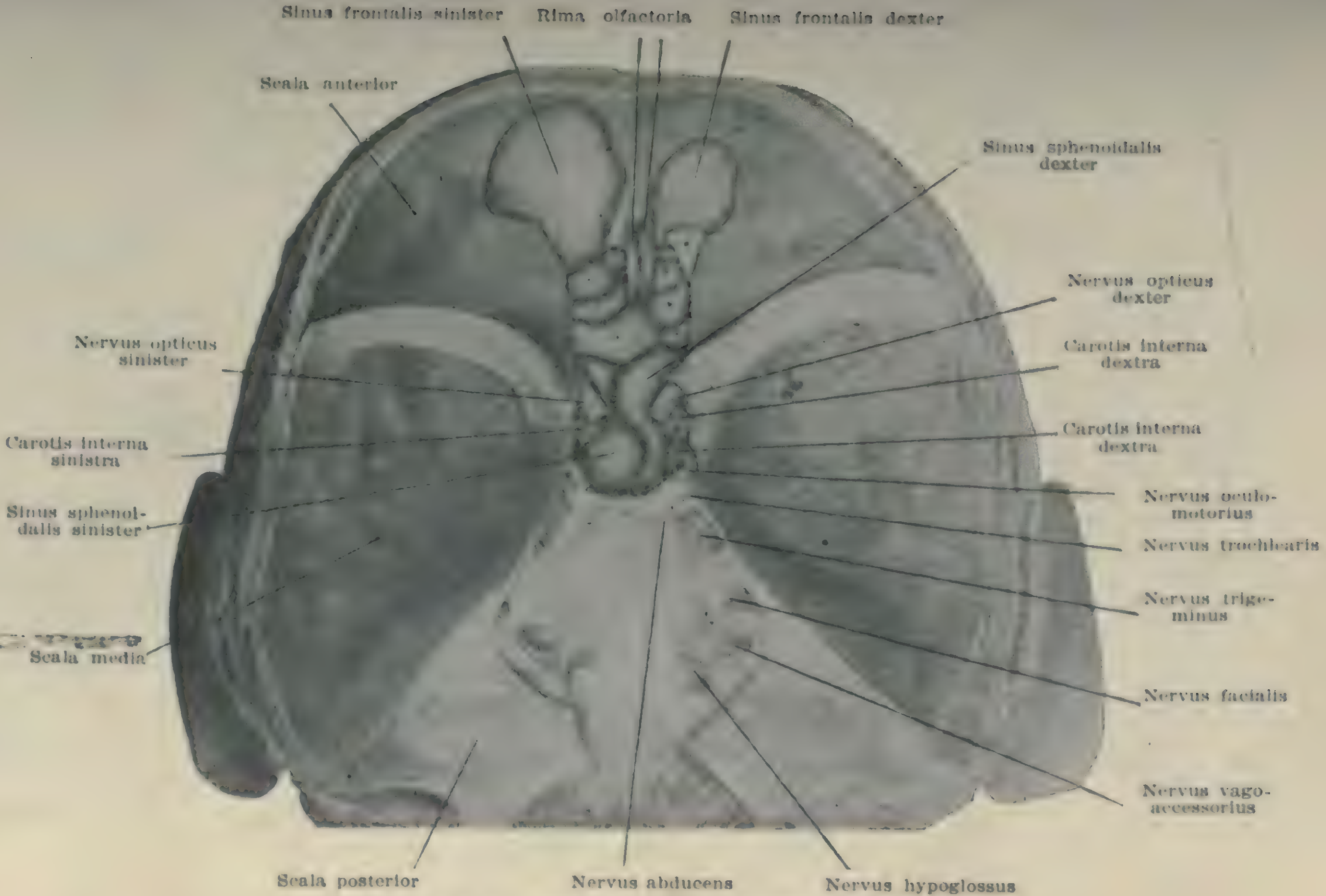

Cellula ethmoidalis posterior dextra
Nervus opticus sinister
Concha superior
Sinus frontalis
Sinus sphe- oidalis nister
Concha media
Ostium naxillar
Meatus narium uperior
Meatus narium medius
Concha inferior
Palatum
Meatus narium inferior

FIGURE 14.



X X X X X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 15.

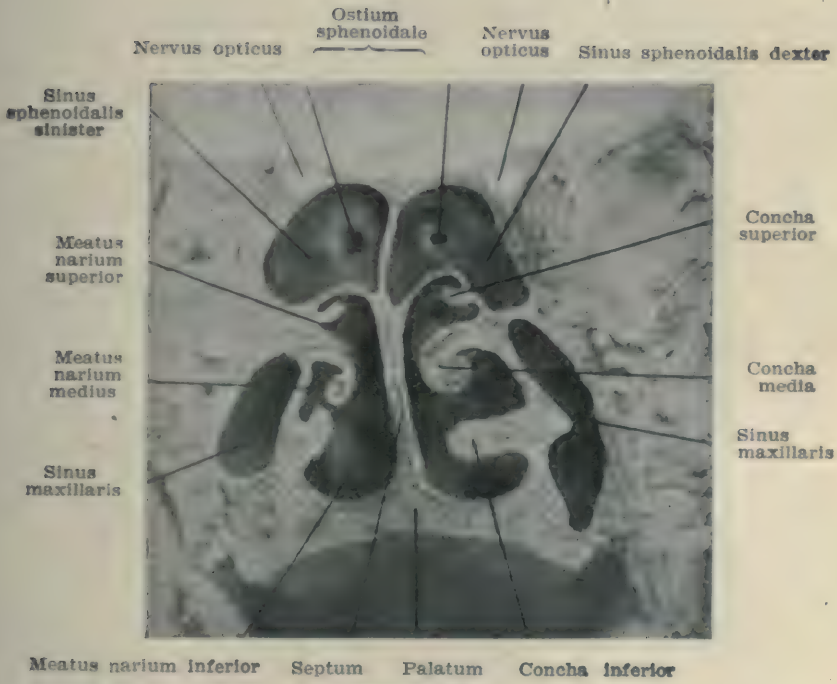

Ostium sphenoidale
Nervus opticus
Nervus opticus
Sinus sphenoidalis dexter
Sinus sphenoidalis sinister
Meatus narium superior
Meatus narium medius
Sinus maxillaris
Concha superior
Concha media
Sinus maxillaris
Meatus narium inferior
Septum
Palatum
Concha inferior

FIGURE 16.

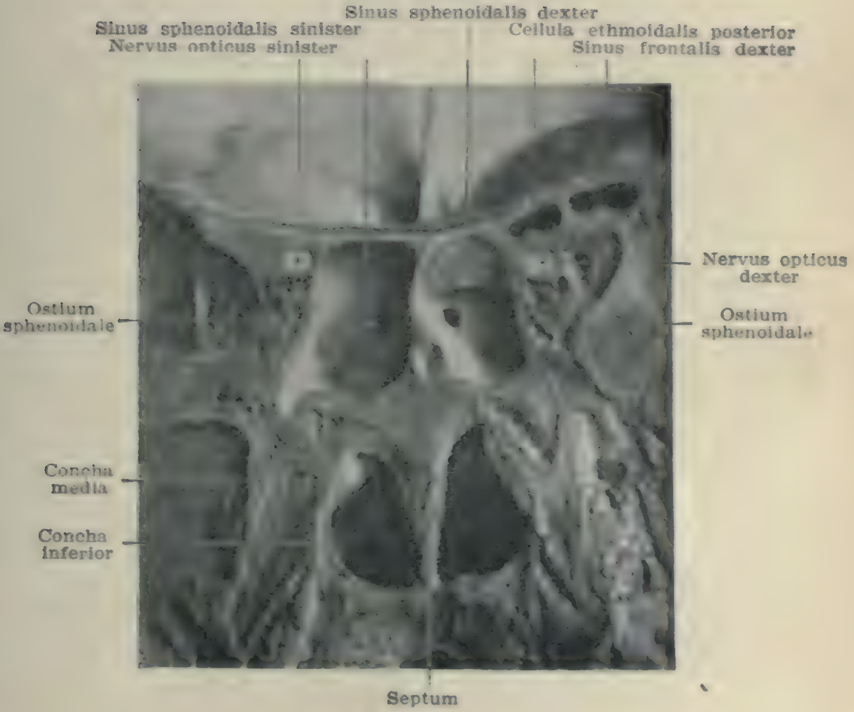

Sinus sphenoidalis dexter
Sinus sphenoidalis sinister
Nervus opticus sinister
Cellula ethmoidalis posterior
Sinus frontalis dexter
Nervus opticus dexter
Ostium sphenoidale
Ostium sphenoidale
Concha media
Concha inferior
Septum

FIGURE 17.

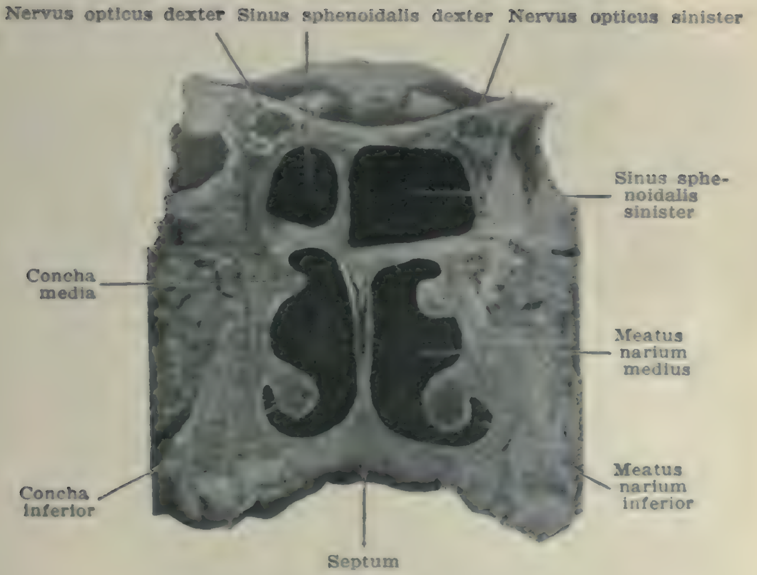
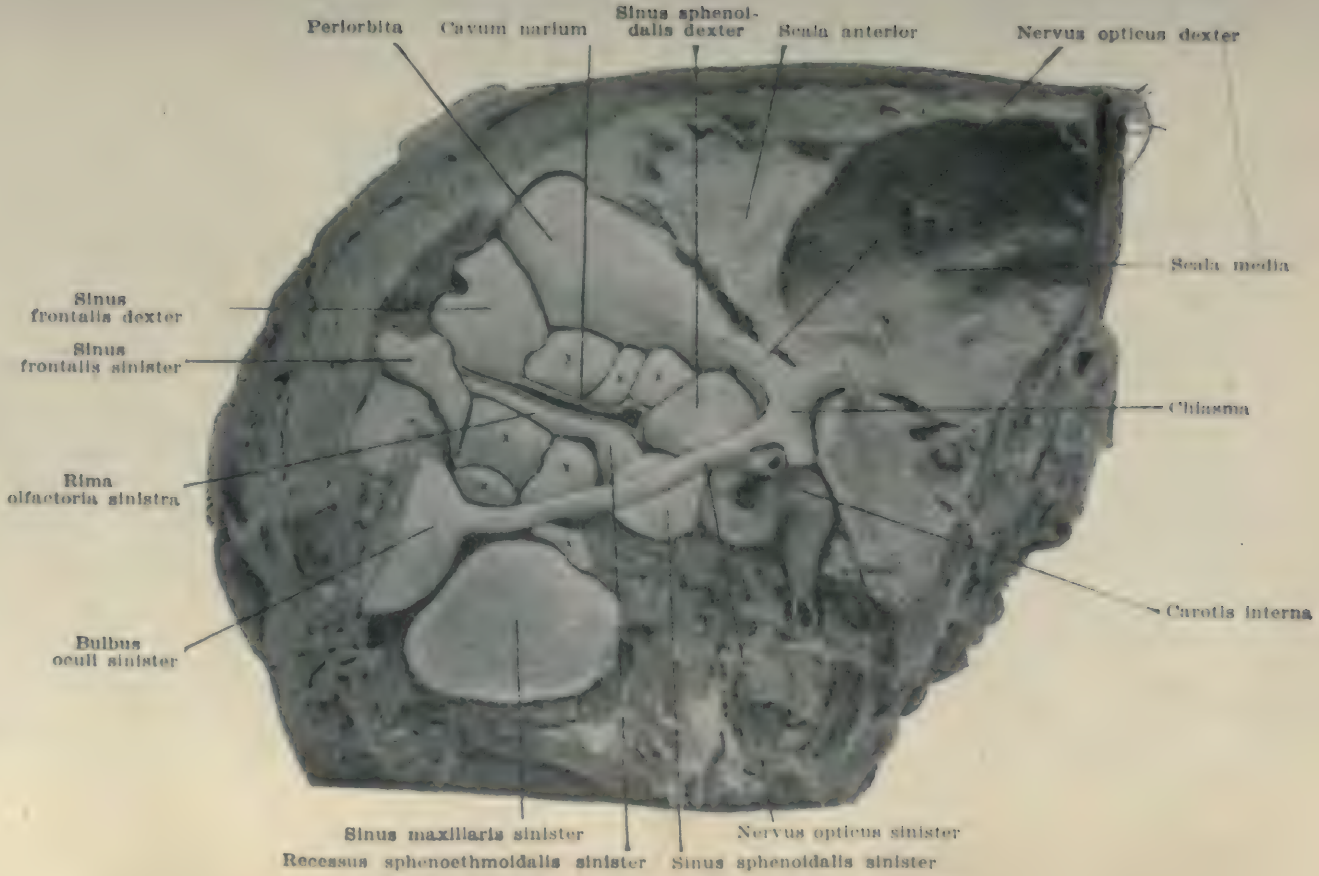

Nervus opticus dexter
Sinus sphenoidalis dexter
Nervus opticus sinister
Sinus sphenoidalis sinister
Concha media
Meatus narium medius
Concha inferior
Meatus narium inferior
Septum

FIGURE 18.



X X X X X X Cellulae ethmoidales anteriores et posteriores

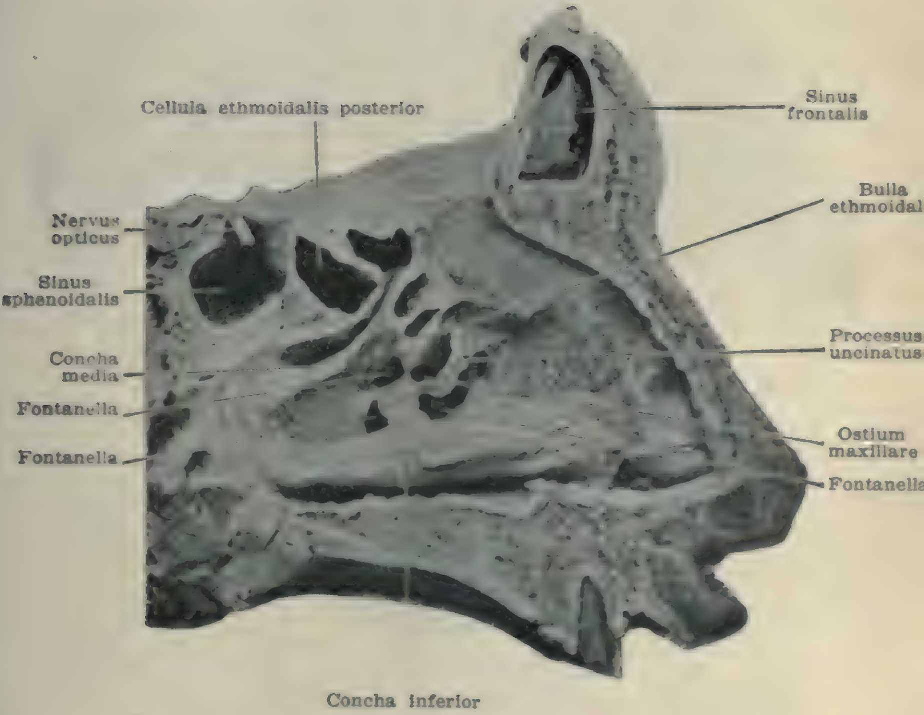
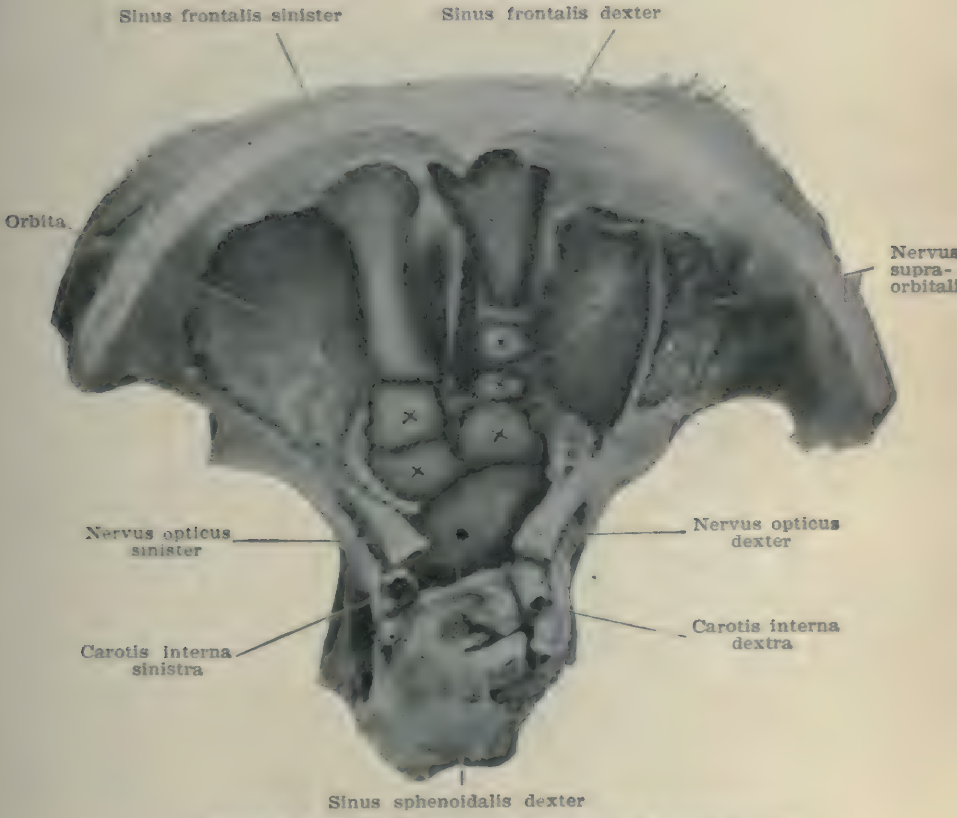

Cellula ethmoidalis posterior
Sinus frontalis
Bulla ethmoidal
Nervus opticus
Sinus sphenoidalis
Processus uncinatus
Concha media
Fontanella
Ostium maxillare
Fontanella
Fontanella
Concha inferior

FIGURE 21.



X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 22.

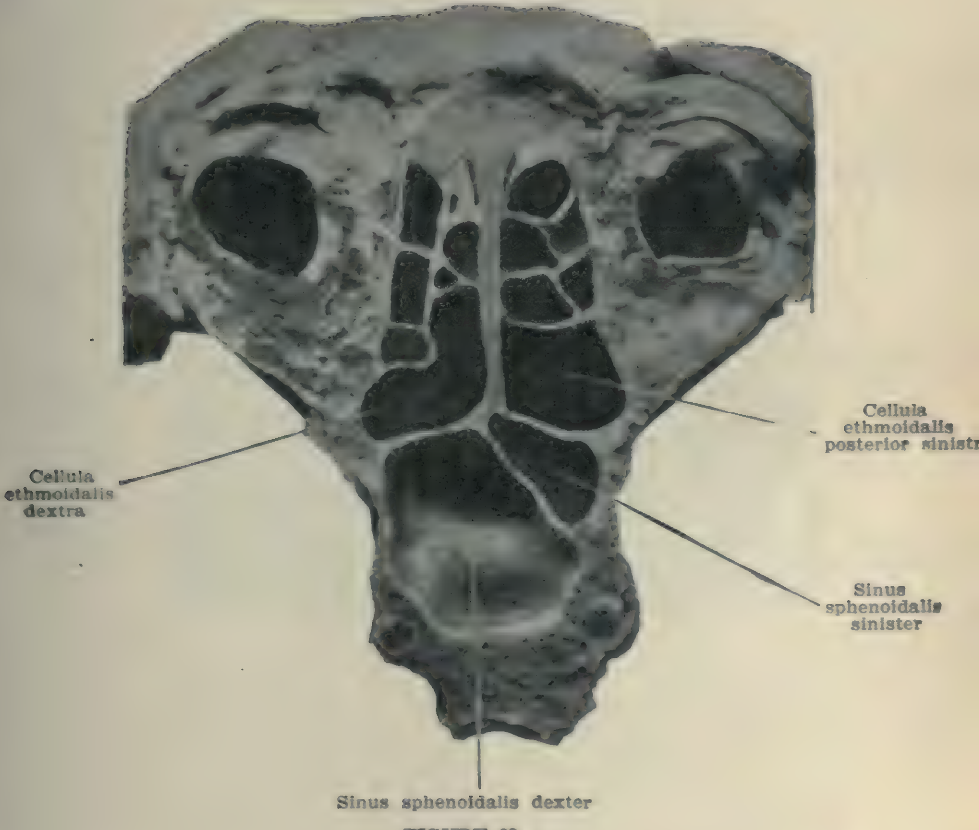

Cellula ethmoidalis posterior sinistr
Cellula ethmoidalis dextra
Sinus sphenoidalis sinister
Sinus sphenoidalis dexter

FIGURE 23.

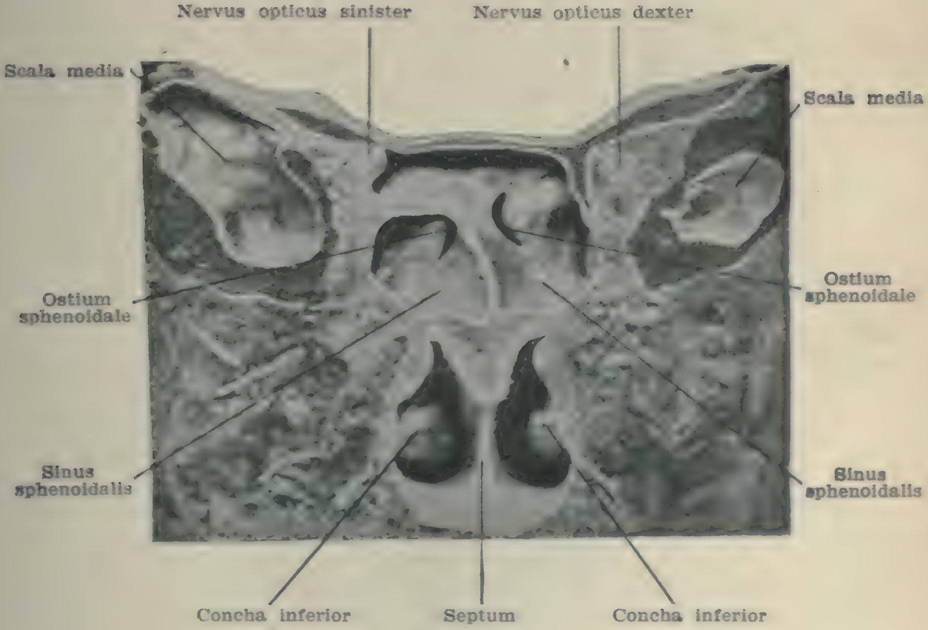

Nervus opticus sinister
Nervus opticus dexter
Scala media
Scala media
Ostium sphenoidale
Ostium sphenoidale
Sinus sphenoidalis
Sinus sphenoidalis
Concha inferior
Septum
Concha inferior

FIGURE 24.

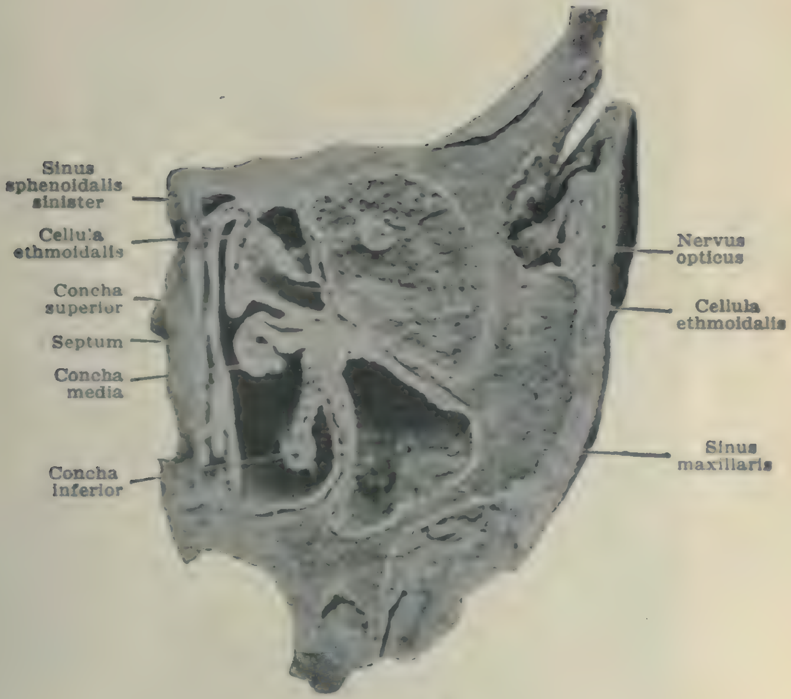
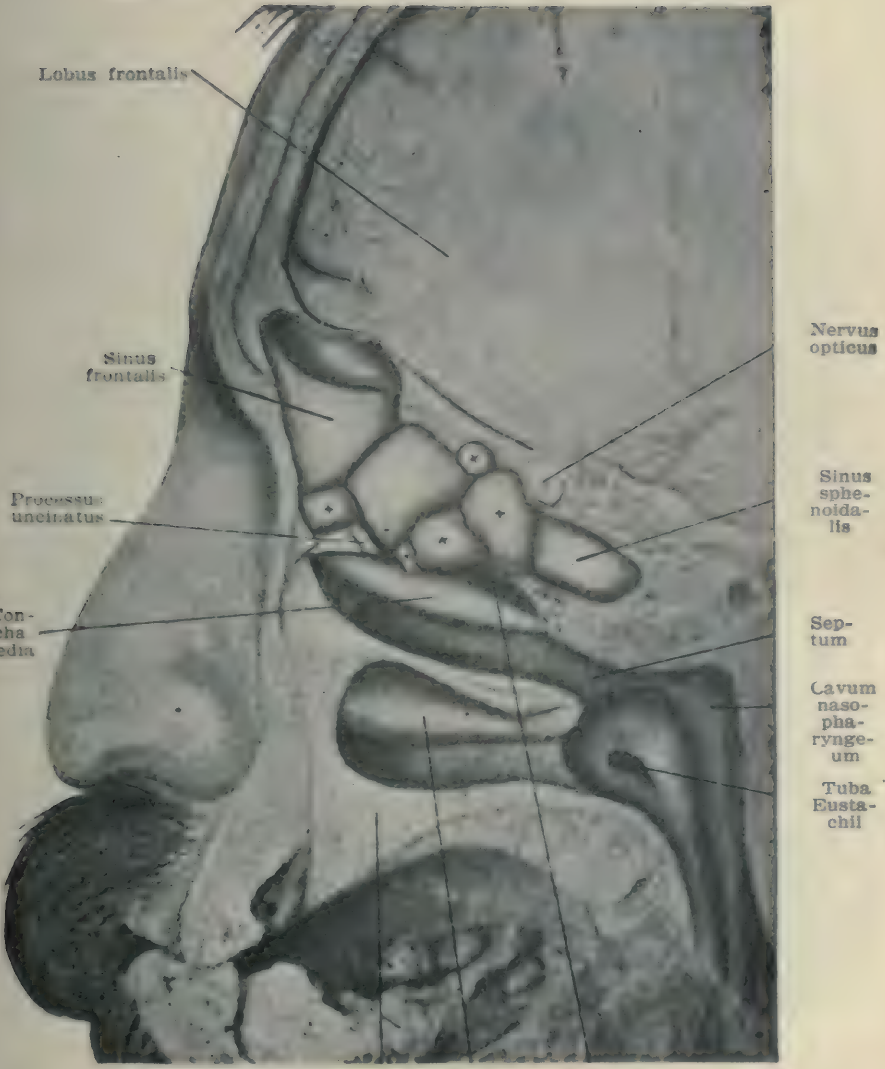
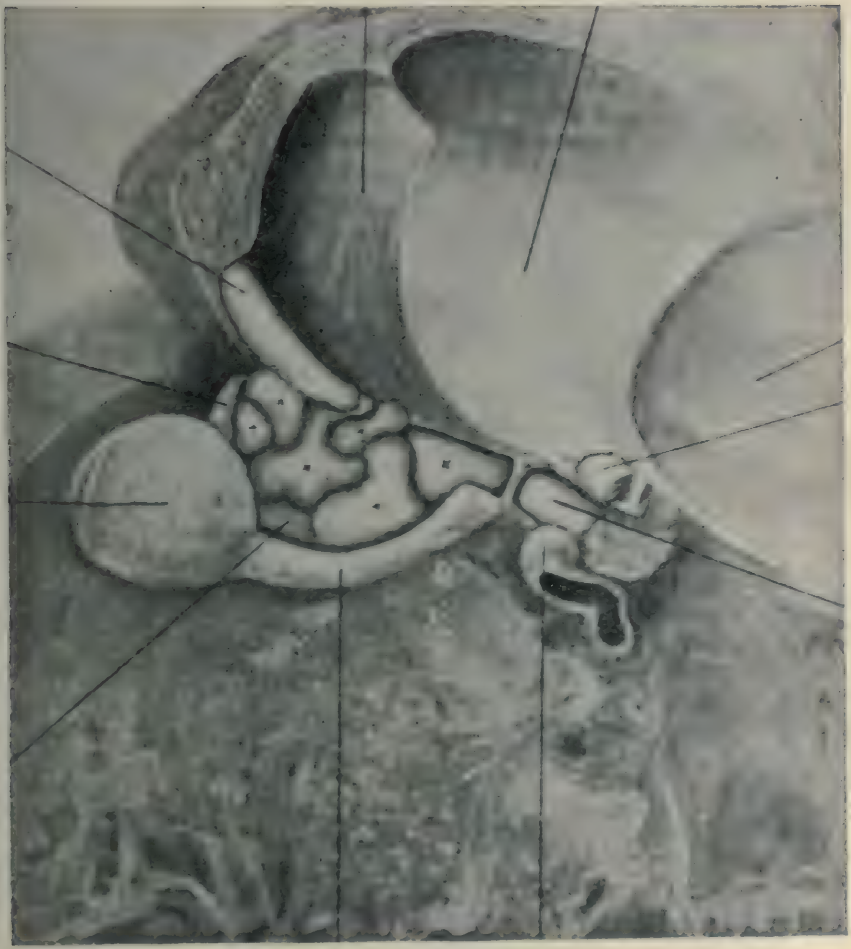

Sinus sphenoidalis sinister
Cellula ethmoidalis
Concha superior
Septum
Concha media
Concha inferior
Nervus opticus
Cellula ethmoidalis
Sinus maxillaris

FIGURE 25.



X X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 26.



X X X X X X Cellulae ethmoidales anteriores et posteriores.

FIGURE 27.

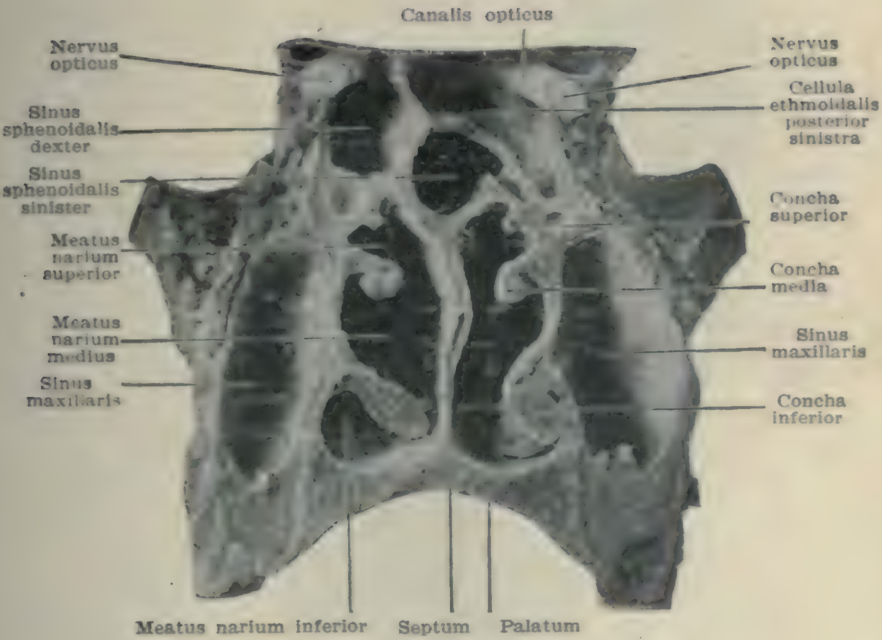

Canalis opticus
Nervus opticus
Nervus opticus
Sinus sphenoidalis dexter
Cellula ethmoidalis posterior sinistra
Sinus sphenoidalis sinister
Concha superior
Meatus narium superior
Concha media
Meatus narium medius
Sinus maxillaris
Sinus maxillaris
Concha inferior
Meatus narium inferior
Septum
Palatum

FIGURE 28.

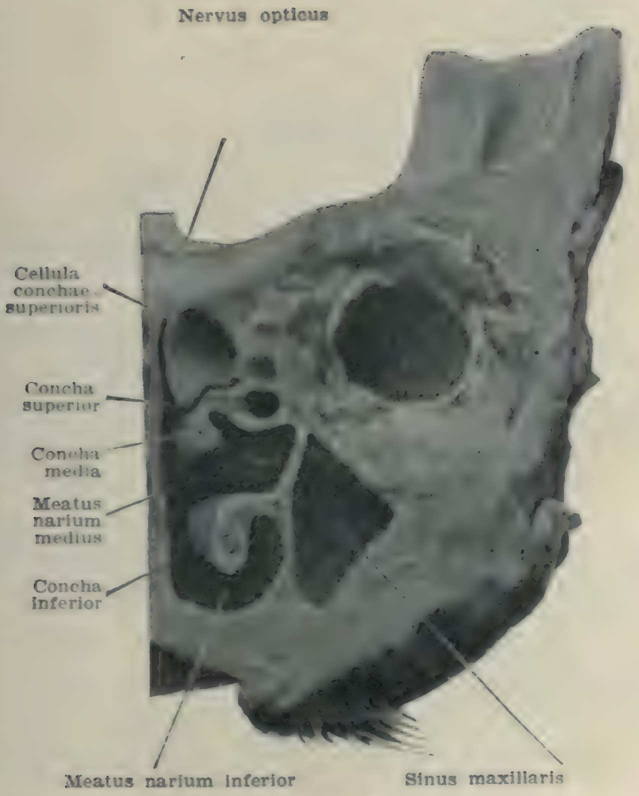

Nervus opticus
Cellula conchae superioris
Concha superior
Concha media
Meatus narium medius
Concha inferior
Meatus narium inferior
Sinus maxillaris

FIGURE 29.

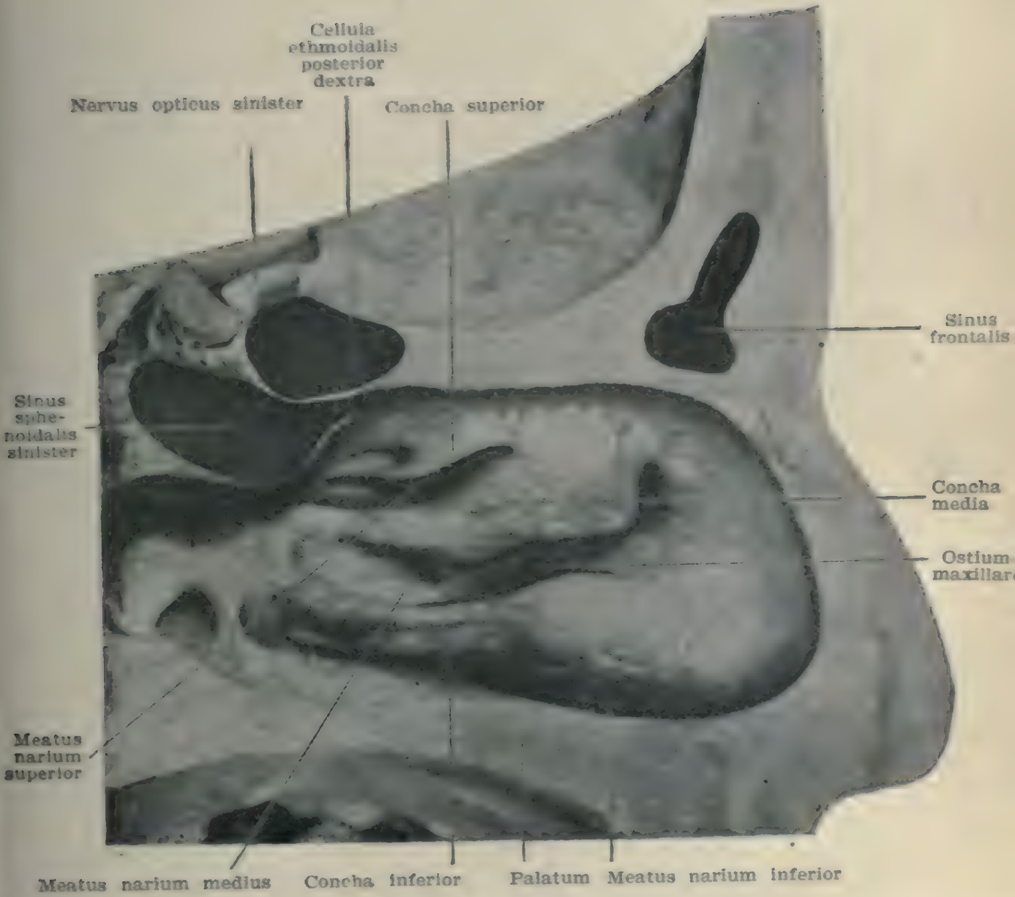
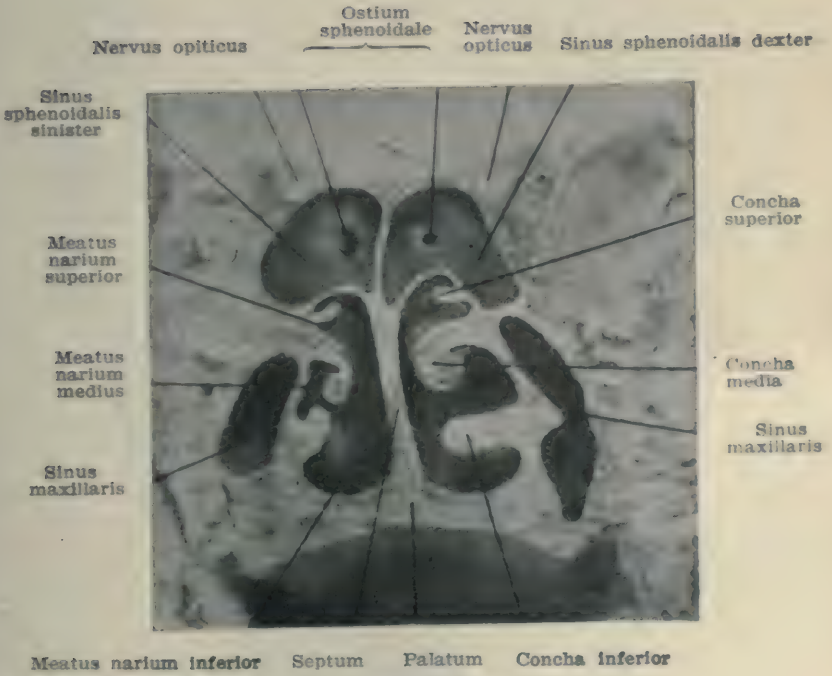

Cellula ethmoidalis posterior dextra
Nervus opticus sinister
Concha superior
Sinus frontalis
Sinus sphenoidalis sinister
Concha media
Ostium maxillar
Meatus narium superior
Meatus narium medius
Concha inferior
Palatum
Meatus narium inferior

FIGURE 30.


Nervus opiticus
Ostium sphenoidale
Nervus opticus
Sinus sphenoidalis dexter
Sinus sphenoidalis sinister
Meatus narium superior
Concha superior
Meatus narium medius
Concha media
Sinus maxillaris
Sinus maxillaris
Meatus narium inferior
Septum
Palatum
Concha inferior

[illegible] FIGURE 31.

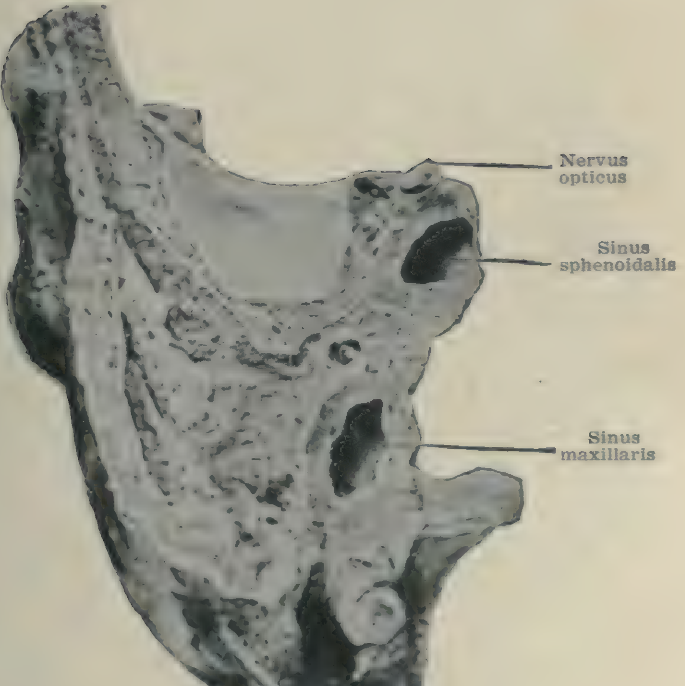


FIGURE 32.

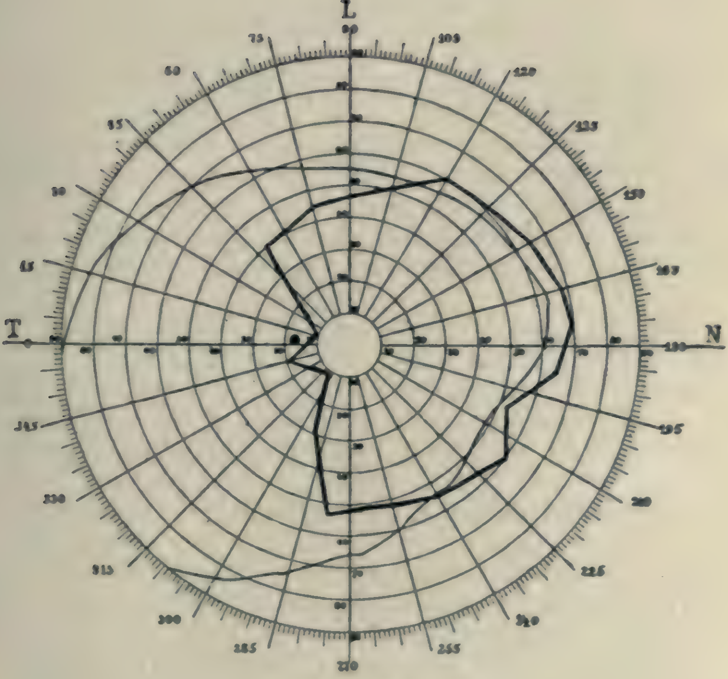

L
90
75
105
60
120
45
135
30
150
15
165
T
N
180
345
195
330
210
315
225
300
240
285
255
270

FIGURE 33.

II.

OBSERVATIONS ON TEN CASES OF INTRACRANIAL COMPLICATIONS OF MIDDLE EAR SUPPURATION.

By S. J. Kopetzky, M. D.,

New York.

The following cases came under observation during the last two years, and because of the variety of the intracranial lesion and the results obtained, are believed of interest. A brief outline of the case histories, covering the absolutely essential details only, has been deemed sufficient, space forbidding an exact repetition of the daily record for each individual case.

Case 1.—F. M., aged 27; came under observation in August, 1906.

She had measles, scarlet fever and whooping cough in childhood. Following a cold three years ago, there developed a discharging ear. For a few days before her appearance at the Manhattan Eye and Ear Hospital, there appeared pain in the right ear and a swelling behind the auricle. Some tenderness upon pressure below the tip was evident.

Diagnosis.—Acute exacerbation of a chronic middle ear suppuration.

Operation.—On August 20th, 1906.

Operative Findings.—Mastoid process completely eburnized. Antrum, when uncapped, found small in size, containing cheesy deposits. Tegmen antri et tympani opened, evacuating extradural abscess. Original opening through tegmen accidentally made. The pus of the abscess is of foul odor. The dura is freely exposed; it evidences pulsation and is covered with dirty granulations.

The operation completed, wound washed and packed; and external dressings applied.

Postoperative Period.—August 21st, patient comfortable. Pulse, 114; respiration, 24; temperature, 100.

August 22nd.—There was nausea and vomiting. Pulse, 124; respiration, 24; temperature, 100.3. The patient complains of feeling dizzy and occasionally as if falling forward. No nystagmus present.

August 23rd.—Wound dressed. The dura appears necrotic, with pus oozing from between dura and bone. The general appearance of the patient seems good, although an increase of nervous irritability is noted. During the day, vomiting spells occurred three times. Pulse, 90; respiration, 22; temperature, 99.

August 25th.—Some horizontal nystagmus noted when eyes are turned toward the healthy side. Later in the day, in addition, vertical nystagmus is apparent. Pulse, 90; respiration, 22; temperature, 100.

Exploratory operation was decided upon because of the persistance of purulent discharge from between bone and dura and because of the apparent breaking down of the exposed dural area, in addition to the continuance of the system's symptoms and nystagmus.

Explorative Operation.—The tegmen was further removed, working medially toward the pyramidal tip and semicircular canals. A small fistulous opening was now revealed in the dura, which, upon further exploration, was found to communicate with an abscess cavity. The cavity was wiped clean and a drain placed in position. Packing of the mastoid wound and external dressings followed.

The patient gradually grew worse, the septic appearance became marked until on August 28th, typical septic temperature was established. Patient was semiconscious and choked discs were present. The dura was examined under the squamosal plate, and the brain cortex found necrotic in areas. This was scraped away. Pus was evident near the tip of petrosal pyramid. These procedures were made under anesthetic. Exploratory puncture failed to locate deep seated abscesses. Death three and one-half hours after exploratory operation.

CASE 2.—J. M., 1 year of age; came under observation March 27th, 1907. The left ear had been discharging for three months. The present illness began with vomiting and loss of weight for some time before the ear discharged.

Simultaneously with the appearance of the otorrhea, a facial paralysis became noticeable.

The patient is much emaciated, semiconscious. The drum membrane is destroyed, the middle ear cavity filled with granulations. Radical mastoid operation performed at the Manhattan Eye, Ear and Throat Hospital, March 28th, 1907.

Operative Findings.—Exposure of cortex revealed a fistulous tract leading directly downward toward the antrum. A second fistulous tract was found spreading backward toward the sigmoid sinus and toward the dura. Both dura and sinus were uncovered and examined. The dura was blanketed with discolored granulations, and at one locality a distinct necrotic ulcerated area was found.

The posterior meatal wall was partly involved in the necrotic lesion, involving the facial nerve whose distal end was imbedded in granulations. The proximal end of the facial nerve was not found. The horizontal semicircular canal wall was necrosed and the canal's lumen open.

Postoperative Period.—At the change of dressing April 1st, 1907, the bandage and dressings were noticeably fouled. The patient was fretful and screamed when moved.

April 2nd.—Muscular spasms of slight nature noted. The child refuses nourishment.

April 3rd.—Dressed. Foul odor in dressings.

April 4th.—Vomiting. Muscular twitching continued. Nystagmus appeared.

April 5th.—Foul odor to dressings continue. Granulations beginning to appear. Slight secretion in external auditory meatus.

April 6th.—Spasms almost continuous. Head and neck rigid and bent back. Nourishment not retained. Fed by rectum, but not retained. Temperature, 100; respiration, 48; pulse, 158.

April 7th.—Temperature, 101; resipration, 38; pulse, 136. Vomiting constantly. Child unconscious.

April 8th.—Temperature, 101.2; respiration, 40; pulse, 156. Jaws locked.

April 9th.—Temperature, 103.6; respiration, 36. General condition unchanged.

April 10th.—Temperature, 104.6; respiration, 24; pulse, 90. Child alternately rigid and relaxed. Nourishment partly retained. Dressing of wound showed dirty. Toward end of day temperature dropped to 102 and again rose to 103.6.

April 11th.—Patient rapidly sinking. Temperature, 104.4. dropping as death comes on.

No second operation was undertaken in this case because of the absolutely poor general condition after the first operation.

The child was brought in semiconscious and never rallied. The radical operation at that time included exposure and exploration of the meninges.

CASE 3.—N. P., aged 1½ years, appeared at the ear clinic of the Manhattan Eye, Ear and Throat Hospital, April 16th, 1907.

Patient had been ill for over a year with gastro-intestinal disturbances. The child was breast fed. For one year there had been discharge from the left ear. For about a week the ear appeared to give pain when touched. For three days the child has had fever. The ear evidenced a profuse otorrhea, with foul odor, the drum presented a large defect through which profuse granulations sprung. The discharge showed a mixed infection. Status on admission, temperature, 101.4; respiration, 56; pulse, 134.

The case was kept under observation for forty-eight hours. On the day following admission, April 17th, the temperature rose to 104, then dropped to 98, rising again to 102.4. Radical operation was performed on April 18th, 1907.

Operative Findings.—Upon retraction of the soft parts, a perforation was found through mastoid cortex, just below the level of antrum. The mastoid cells were filled with pus, granulations and detritus. The tip was removed. Inner table of mastoid was outlined. Postmeatal wall was lowered, connecting the tympanic cavity with the antral opening. Low wall of the meatus was found necrotic and was partially removed. Eustachian tube curetted. The horizontal semicircular canal wall was found eroded. Wound cleansed, packed and dressed.

Postoperative Period.—Following the radical operation, the child did well, from April 19th to April 23rd. No abnormal temperature rise noted. On April 23rd, Panse flap was cut, placed in position, postauricular wound sutured and the dressings reapplied.

April 24th.—Beginning facial paralysis becomes evident. The temperature rose late in the day to 100.6; respiration, 52. pulse, 150. A rash appeared covering the entire body. Thinking it perhaps scarlatina (for it looked so), a diagnostician from the health department was called in, who declared the rash non-contagious. Some cough developed and on April 24th, the temperature rose to 103.8; pulse, 159, respiration, 50.

Two small areas of consolidation became evident in the lungs, in the postscapular region.

On April 25th, the rash is still present. The case was seen by attending pediatrician and pronounced non-contagious. The rash appeared like small petechial spots.

Dressing changes meanwhile showed mastoid wound to look healthy. Facial paralysis still present.

April 26th.—Temperature fell, gradually reaching normal. The pulse rate and respiration dropped proportionately. The rash is still present on face and body.

The child improved and was finally discharged from the wards and sent to the out-patient department. May 2nd, still under personal observation. Facial paralysis complete.

From May 2nd to 11th I saw and dressed the wound in the out-patient department.

May 11th.—Child was brought in with temperature of 103; pulse, 128; respiration, 28. Child refused nourishment, is restless and semiconscious, and head slightly drawn back. Smear from wound gave streptococcus infection. Immediate exploratory operation.

Operative Findings.—The old mastoid wound cleaned out. Incision lengthened upward. Periosteum retracted. Squamosal plate opened, dura exposed. No pus found. Dura opened. Brain found normal.

Posterior and superior semicircular canals opened. The promontory opened and the vestibule and cochlear whorl cleansed. Pus was found in the internal ear channels. The stapes was imbedded in a mass of granulations. During the manipulation about the stapes nystagmus was absent.

Skull cortex behind the mastoid wound was opened, dura over cerebellum discolored. Incision released discolored and purulent cerebrospinal fluid under pressure.

A lumbar puncture made at the time of operation gave only 8 cc. of cloudy fluid. This fluid showed 71 per cent polynuclear leucocytes, and 29 per cent of lymphocytes. Smears from the puncture fluid gave a small number of streptococcus chains.

The child's condition was bad immediately after operation and suitable stimulant restoratives were administered.

May 12th.—Temperature ranged from 103 to 104; the pulse from 150 to 160 and respiration from 44 to 78. Generally, the condition was unchanged.

May 13th.—A second lumbar puncture was made. The fluid was thick and tinted. Not found under pressure and only about 8 cc. withdrawn.

May 15th.—Lumbar puncture obtained only two drops of a very thick fluid. Temperature between 103 and 104; respiration, 54-60; pulse, 140-168. The general condition worse. Death, May 18, 1907. Temperature, 103; respiration, 98; pulse, 180.

After each puncture, and especially after the escape of fluid upon incision of the cerebellar dura, the rigidity of the head and neck disappeared, and the child appeared brighter. The failure to obtain fluid at puncture under pressure, when it was shown in the subarachnoid space under high tension, is accounted for by a closure of the foramen of Magendie.

Case 4.—F. T., aged 15 months, came under personal observation, giving the following history:

For about a week the child suffered from gastro-intestinal disturbances and loss of weight. Child had had fever. Both ears were suspected because of evident distress when they were handled. Paracentesis was performed on both ears, June 10, after which otorrhea became established. Mastoid process was tender to touch and some swelling was noticeable behind the ears.

Expectant treatment was continued till June 13th, when child was admitted to the hospital, with temperature, 102; respiration, 20; pulse, 140, and simple mastoidectomies were performed on both sides.

Operative Findings.—Retraction of soft parts showed an intact cortex. Antri opened and filled with pus. Mastoid cells eviscerated. No sinus or dura was exposed.

The wound cavities were irrigated with normal salt solution, permitted to fill with blood and subsequently treated by the blood clot aftertreatment, subcutaneous sutures being used.

The temperature dropped to normal upon the second day after operation and remained normal thereafter.

June 16th.—A slight odor noticeable in the dressings from the right ear, some serous oozing from the lower wound end. The odor continued to be noticed, and finally on June 17, 1907, also appearing on the left side, the wounds were opened, the cavities washed with antiseptics and subsequently dressed with the usual open method.

Thereafter the case continued uneventful, the granulations gradually filling in the cavity, and the wounds on both sides becoming fully healed.

July 10th.—Both ears being examined and found dry, the case was discharged. Control examination, July 20th.

For about one month nothing was seen of the patient.

August 10th.—The child awoke during the night, as reported by the mother, with a facial paralysis on the right side. The child vomited during the night a number of times, and the following morning was readmitted to the hospital. Temperature, 103; respiration, 28; pulse, 130; facial paralysis and otorrhea on right side.

That afternoon muscular twitchings noted in hands and arms. Pupils dilated. Head drawn backward. Some convulsions immediately before operation. Temperature at time of operation, 103; respiration, 50; pulse, 130.

Exploratory Operation.—August 12, 1907.

Operative Findings.—Incision along old scar on right side. Periosteum retracted, and radical mastoid operation performed. Necrotic bone discovered in antral region, and over inner tablet of mastoid covering middle cranial fossa. The postmeatal wall lowered. Tympanic cavity cleansed, ossicles removed. Anterior mastoid plate lowered. The stapes found imbedded in granulations, with perforation of its annular ligament. The labyrinthine windows were opened and connected, cochlea opened, and horizontal semicircular canal opened. The vestibule was cleansed. Facial nerve was exposed. Dura was exposed and found healthy in appearance. Pressure on jugulars caused increased flow from opening in semicircular canal.

Following the operation, more convulsions noted. All limbs involved. At midnight, vomiting and extreme restlessness.

The following morning temperature, 104.2; respiration, 36; pulse, 140. Later respiration spasmodic, pulse weak.

A difference in the size of the pupils noted. Right pupil larger than left. Death soon followed.

Case 5.—A. R., aged 18 years, was admitted May 11, 1907, giving the following history. Had typhoid-malaria several years ago. Never had any of the exanthemata.

Left ear has been discharging on and off since infancy whenever patient caught a "cold."

Her present illness began three weeks ago, with pain in the ear, and discharge.

Examination showed absence of edema about pinna. Marked pain on pressure over tip and lower portion of mastoid generally. Otoscopic examination revealed perforation in lower posterior quadrant, discharging thin purulent matter. No sagging of canal walls, no headache, twitching of muscles or vomiting. General appearance septic. On admission, temperature, 100; respiration, 20; pulse, 92. Immediate operation was deferred because patient seemed comfortable and urine showed marked albuminuria with red and white cells, granular and hyaline, and epithelial casts.

Ear smear showed a few extracellular diplococci.

Patient was put upon a milk diet, diuretic administered and the ear locally treated.

May 12th.—Morning temperature showed 100.4; respiration, 24; pulse, 96. In the afternoon patient had a chill and temperature rose to 105.4; respiration, 22; pulse, 126. A foul smelling otorrhea followed paracentesis. Immediate operation was ordered.

Operative Findings.—Mastoid found eburnated. Cortex opened and pus located in a tract leading from tympanic cavity to the sigmoid sinus whose walls were found opened and whose lumen contained fluid pus. No bleeding obtained from the sinus. The inner tablet was removed backward, to within one-half inch of the torcular before blood was found. From bulbar end no blood was obtained.

Without curetting bulb and avoiding all unnecessary manipulations, the internal jugular vein was laid free, tied off about one inch above the clavicle and resected to the point of entrance of the facial vein. Then the jugular bulb was curetted.

The wounds were cleansed, packed and dressed. Purse string sutures were used on the neck wound.

Postoperative Period.—Following operation, the temperature promptly fell to 100.4; respiration, 26; pulse, 124.

May 13th the urinalysis showed albumin negative, with absence of red, white and epithelial cells. A few hyaline, granular casts were still present.

May 14th.—Temperature, 98.4; respiration, 24; pulse, 98. Patient generally comfortable.

Dressing changes showed lower wound perfectly clean. String sutures drawn together and lower part of wound closed. Mastoid wound clean. Plug changed over torcular end of sinus. From May 14th to May 19th, temperature ranged from normal to 99.8, lower in the morning than in the evening. The pulse ranged from 88 to 96. The respiration from 20 to 22.

In general, conditions seemed so favorable that the patient was permitted to get out of bed.

May 21st.—Headache was complained of.

May 20th to 22nd.—Temperature about 100; pulse 80 to 90; respiration, 20 to 22. The dressings changed May 22nd gave an offensive odor from upper wound. Appearance healthy, however. Lower wound (neck) clean, middle ear somewhat dirty looking.

Patient was put to bed because of occiptal headache, some nausea and vomiting. Temperature, 100.6 to 102.6; respiration 22 to 24; pulse, 96 to 110.

May 23rd.—Eyes examined. Slight blurring of discs on temporal side. Some lateral nystagmus.

No dizziness, patient stands quietly and does not stagger when walking.

May 24th.—Lowest temperature, 99.6; highest, 101.4; respiration, 20; pulse, 96 to 106. Patient appears to be drowsy.

May 25th.—Dressed. Very foul odor present in posterior part of mastoid wound. Some pus is evident at upper part of neck wound. Temperature ranged between 101 to 101.4; pulse, 100 to 104; respiration, 22.

After consultation with Dr. Wendell C. Phillips, exploratory operation was undertaken.

Exploratory Operation.—Posterior wound extended backward. Cortex further removed. Visceral side of sinus wall examined and found healthy. Dura over cerebellum examined and found healthy. Bulb examined and found free from pus. Irrigation of wound, and packing replaced.

May 26th.—Temperature, 101.4 to 100; respiration, 20 to 22; pulse, 98 to 90. Patient vomiting frequently.

May 27th.—No change.

May 28th.—Patient very drowsy. Occipital headache. Gradually became weaker, and comatose.

May 29th.—Vertical nystagmus noted. Left pupil larger than right. Right fundus and disc blurred.

May 30th.—Death.

Postmortem examination of mastoid wound gave pus behind visceral wall of sigmoid sinus, extending toward cerebellum; extent not determinable as autopsy was refused.

CASE 6.—(Seen in association with Dr. R. Johnson Held).

Male, aged 52 years, had suffered from chronic otorrhea for three years. For six months previously had indefinite pains over mastoid region. Three weeks ago facial paralysis came on.

Examination showed all symptoms of mastoiditis on the right side, with facial paralysis. Temperature, 101; pulse, 92; respiration, 22. The patient seems indifferent to his condition, exhibiting retarded cerebration. There was marked tenderness over entire mastoid. Considerable thick discharge from the ear, greenish in color and of a foul odor. Tympanic cavity was full of granulations, making the membrana tympani invisible.

Radical operation performed April 24, 1906.

Operative Findings.—Upon removal of cortex, considerable pus, granulations and bone detritus were uncovered. The entire cellular character of the mastoid had been destroyed under the purulent attack. The internal mastoid table was absent, exposing the sinus and cerebellar dura. These were covered with discolored granulations. The tegmen aditus ad antrum was absent. The postmeatal wall was destroyed, including the major part of the external (lateral) aditus wall. In other words, "Nature had attempted the radical."

After cleansing, examination revealed absence of the tympanic floor, the jugular dome being free in the contents of the tympanic cavity. The facial nerve was found exposed, diseased and imbedded in granulations. The bony framework of the Eustachian tube had disappeared, as well as the anterior tympanic wall, exposing the capsule of the joint of the lower jaw at the glenoid fossa.

The dura over aditus and tympanic cavity was found perforated, pus exuding. Upon exposure of the brain substance, a sloughing ulcer of the brain surface was uncovered, 1.5 cm. in area, 0.5 cm. in depth.

The wound was cleansed, packed and dressed. The patient made a complete recovery. Facial paralysis persisted. Upon inquiry, I learned that he died of some intercurrent disease one year later.

During the entire postoperative period the temperature never rose above 100.

Case 7.—(Seen in association with Dr. R. Johnson Held and reported by us in the *Archives of Otology*, Vol. XXXV, No. 6, 1906; republished here to complete histories of material from which paper was written). R. R., aged 3 years, appeared at the Manhattan Eye, Ear and Throat Hospital, December 13, 1904, suffering with a chronic middle ear suppuration. The radical operation was performed April 29, 1905. At this operation, necrosis of bone was found, extending up to the dura, over the tegmen. The left ear was operated upon March 13, 1906, the radical mastoid operation being performed. In this operation the dura was exposed because bone necrosis extended to and involved the tegmen. The dura was found covered with dirty granulations.

On May 8th symptoms of meningitis came on. The head was markedly retracted, the pupils dilated, the right side deviated outward. The patient was restless and cried continuously. Temperature, 102.4; pulse, 144; respiration, 75. Examination revealed pus in the wound cavity.

Exploratory operation was performed May 12, 1906, the patient having been in a condition of stupor and unconsciousness for twenty-four hours.

Operation.—The old mastoid wound was reopened and cleansed, all granulations were removed, the dural surface exposed and discolored granulations upon it cleaned away.

The lower part of the squamosal plate of the temporal bone was opened and a considerable extent of its area (3x3½ cm.) removed. Through this, the dark congested dura bulged. The dura was incised by horizontal incision, extending the entire length of the exposure. From this incision purulent fluid escaped. Brain substance immediately protruded into the wound. The brain tissue was extremely friable. The wound was flushed with hot saline solution and the entire surface carefully wiped. The brain was carefully lifted, and wiped underneath, and a strip of gauze introduced between brain and dura downward and inward toward the interior of the skull. The gauze was kept in one long strip, the end of which protruded into the external dressings. The mastoid wound was then flushed, packed and dressed. Temperature chart on the next day showed: Temperature, 105; pulse, 170; respiration, 80.

May 14th—The left lateral ventricle was punctured and one and a half drams of purulent fluid withdrawn. Lumbar puncture showed 40 cc. of grayish-white turbid fluid. Following these measures, an improvement was noted. Temperature ranged from 101 to 103.2; respiration, 24 to 30; pulse, 120 to 140.

May 16th.—Lumbar puncture was again performed and 30 cc. of turbid fluid withdrawn. Temperature chart gives temperature, 99 to 101.8; respiration, 26 to 30; pulse, 100 to 140. There is less restlessness, and patient had natural sleep.

May 18th.—Lumbar puncture gave 35 cc. of clear limpid fluid. This had some white sediment, some blood, but no coagula.

Decided improvement in the general condition. Less retraction of head, less restlessness, etc.

The first dressing was made on May 23rd. The wound appeared healthy and was covered with clean, healthy granulations. The dura still appeared thickened but of lighter color and only slightly congested. Temperature, 100; pulse, 106 to 130; respiration, 24 to 30.

From this on improvement continued. The intelligence cleared, temperature became normal, the rigidity of the back disappeared, the eye fundus clear, and case was finally discharged cured.*

Case 8.—A. K., aged 16 months, came under observation October, 1907. The child had been sick about two weeks, with indefinite symptoms, some fever, gastro-intestinal disturbances, etc. For the last few days a swelling had been apparent behind the left ear. The swelling presents the appearance of subperiosteal abscess. The tympanic membrane was not perforated, and extremely bulging.

On October 27, 1907, simple mastoidectomy was performed.

Operative Findings.—Swelling over the cortex upon incision failed to evacuate pus. The tissues were indurated. The cortex upon retraction of the soft parts showed a perforation, the fistulous tract leading directly downward toward the antrum. Fluid pus was evacuated, coming away in pulsations. As the mastoid cortex was removed backward, the sinus wall was uncovered, liberating an accumulation of pus between its dural walls and the overlying bone. The sinus was coated with granulations. The tegmen was removed to evacuate pus extradurally.

The case made an uneventful recovery, until the fourth week of the after-treatment, when fever reappeared. The granulations were curetted and cleaned, and following this the case recovered.

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*The merest mention of the principal findings of this case are here given, as a full and detailed report has already been published, as stated above

Case 9.— A. H. E., aged 38 years, came under observation September 4, 1907. Following a cold, pain appeared in the right ear. Paracentesis was performed, and under local treatment by his physician, he apparently recovered, a discharge from his ear persisting, however.

After an interval of a week, the pain reappeared and the patient then came under my observation.

The right ear evidenced a small perforation, rather high up, from which pus came away under pressure. Paracentesis was repeated. Mastoid tenderness was evident only over the tip. There was no rise in temperature, and the patient seemed comfortable.

The pain gradually became more marked, and a swelling appeared over the mastoid, especially marked at the tip. This gradually spread upward, the edematous swelling finally reaching the upper pole of the auricular attachment.

On the drum, no marked bulging was evident, nor was there any sinking of the posterior superior wall

Simple mastoidectomy was decided upon and the operation was performed September 25, 1907.

Operative Findings.—The incision into the postauricular swelling failed to evacuate fluid pus. The tissue was extremely edematous and indurated.

The mastoid was found to be broad from before backwards and unusually low from above downwards. Its cortex was very irregular, and the posterior root of the zygoma was very marked, stretching backward as a hard ridge of bone. The petromastoid was unusually well marked for an adult.

The retracted cortex evidenced a perforation, ¾x½ inch, situated 1 inch above the broad flat mastoid tip. A probe introduced into this could be passed upward into the antrum and passed backward, pressing against soft granulation on the exposed sinus. The interior was filled with a blood-tinged pus.

The removal of the cortex, showed the entire mastoid process to have been disintegrated and filled with granulations and detritus. The mastoid process was only partly pneumatic. The tip was removed, the posterior meatal wall was found undermined by the purulent process and the facial ridge slightly involved. Of the posterior wall, as much as possible was removed, leaving only a thin ridge to support the external auditory canal.

The zygomatic cells were eviscerated, and the cells in the angle between the tegmen above and the sinus knee eviscerated.

The exposed area of the sinus was examined, cleansed of all pus, the granulations being left undisturbed. Further exposure of the sinus wall was made until healthy tissue was uncovered.

This case made an uneventful recovery.

CASE 10.—J. C, aged 22 years, came under observation October 10, 1907, giving a history of having had a cold, followed by pains in the right ear, four weeks previously. The ear has discharged profusely ever since. The patient has had continuous pain in the ear, and pain on pressure, with a fever for most of this time. Operative treatment had been repeatly advised and had been persistently refused. For two weeks there have been frequent dizzy spells with nausea and vomiting. Lately the pain has subsided, but the dizziness, nausea and vomiting continue, and the patient is feverish, unable to take food, and so weak that she cannot stand unsupported. The general appearance evidences profound sepsis. Temperature, 103.8; pulse, 132; respiration 30. The external ear is full of foul-smelling pus, making a view of the drum impossible.

The ear stands off from the head, giving a postauricular swelling the appearance of a subperiosteal abscess. The greatest amount of induration is noted near the upper pole of the auricular attachment. Immediate operation recommended.

Operative Findings.—The incision into the postauricular swelling failed to show any pus present. The soft parts were found edematous and friable.

Retraction of the soft parts demonstrated an intact cortex. The first opening into the cortex evidenced pus coming away under extreme pressure. The entire cortex removed. Fluid pus and detritus eviscerated. The evisceration of the mastoid contents followed. Pus was traced to the tip which, when removed, revealed an abscess formation in the neck tissues. The cells spread far backward. Breaks in contiguity in the inner mastoid table showed pus behind this. The inner table was removed, uncovering an extradural abscess over the cerebellar dura. This was evacuated. The sinus was exposed, and because of its apparently unhealthy condition, plugged off, opened and examined. No thrombus was found.

The patient being in so precarious a condition, that anesthetic had to be suspended to administer restoratives, further procedures were curtailed to the utmost essentials, and after drainage of the antrum was established, further operative measures were suspended.

The next day the patient's condition was so precarious that it was not deemed advisable to give further anesthetic. The third day, however, her general condition was such that we deemed it advisable to proceed. Further procedures were deemed necessary, in view of an increasing somnolence and stupor and a beginning rigidity of the neck.

The Exploratory Operation.—The tegmen antri et cellulae was opened to liberate a large quantity of pus between dura and bone. The radical mastoid operation was then finished. The dura was incised to afford intrameningeal drainage.

The patient did not recover from the general profound sepsis, and during the next twenty-four hours died.*

The youngest of these ten patients was 12 months, the oldest 52 years of age. The sexes were represented by six females and four males. The process was located on the right side in six cases, and on the left side in four cases. The ear disease had existed from two to four weeks in the acute cases; from 18 months to 17 years, in the chronic cases, before coming under treatment.

The acute conditions notably concerned children. A certain interval was noted between the first operation and the onset of the intracranial lesion. In two of these cases, the ear complication developing under sudden facial paralysis and a high fever. Another case was characterized by gastro-intestinal disturbances, and the absence of a discharge from the ear, attention being finally directed to the affected ear by the onset of otorrhea, together with the manifestations of facial parlysis. While the internal ear was involved in the intermediate lesion in these three cases, this extension of the pathologic process could be averted in another case, also concerning a child.

All the external evidences of subperiosteal abscess were present in three cases, but no fluid pus escaped from the indurated and edematous tissues overlying the mastoid process, after the skin had been incised. In four instances, the mastoid cortex was found perforated (three children; 1 adult, aged 38 years).

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*Since paper has been in preparation, another case has come under observation, a child 10 months of age, with serous meningitis following double mastoidectomy (acute case). Meninges were opened and drained,—patient is recovering. Full report of this case will be given later, as it presents special features.

There was inequality of the pupils in certain cases, the larger pupil in two instances corresponding with the diseased side. One case of sinus thrombosis showed marked albuminuria, which disappeared after the evacuation of the clot.

Five times the middle cranial fossa was invaded; four times the posterior cranial fossa alone; twice, both the middle and posterior cranial fossa.

The pathologic process, in a general way, advanced along two avenues: (1) The direct route, illustrated by seven cases, in which the infection traveled by contiguity from the middle ear to the inner table covering blood vessels or tegmen dura, with subsequent involvement of the meninges. (2) The indirect route, illustrated by three cases, in which the infection of the middle ear did not reach the meninges and brain until after preliminary invasion of the labyrinthine channels.

PATHOLOGIC LESIONS IN THESE CASES.

	Cases.
Pachymeningitis externa (extradural and perisinus abscess)	7
Pachymeningitis interna (intradural abscess, cerebral and cerebellar)	4
Cerebral abscess (temporosphenoidal lobe)	1
Cerebellar abscess	1
Sinus thrombosis (involving jugular bulb)	1
Ulcerations on the brain cortex	1
Purulent labyrinthitis	3
Pus in the lateral ventricle	1

COMMENTS ON THE LESIONS.

The most frequent intracranial lesions of otitic origin consist in purulent inflammation of the external surface of the meninges and the formation of extradural abscess. As a rule, the process is referable to bone necrosis involving the dura, or it may develop in connection with caries or cholesteatomatous degeneration of the temporal bone. In the majority of the cases, the disease can be traced to the dura. A collection of pus in the cerebellar region appears in shape of a perisinus abscess. Although generally located over the tegmen, when the middle cranial fossa is invaded, the pus has been known to occur at the top of the petrosal pyramid.

Bony disintegration is not necessarily complete, in abscesses complicating suppurative middle ear disease. Not infrequently, there is partial eburnization of the bone, with purulent tracts burrowing through it. The dura at the site of the abscess presents discoloration, and may be covered with a layer of granulations. Superficial ulceration, with purulent infiltration, has been noted on the meningeal membrane, which in other cases appears thickened and raised up from the bone, while a thick stream of fetid pus oozes between the meninges and the bone.

These variable findings I am inclined to interpret as pathologic stages rather than as types; the time at which the disease is first noticed representing an essential factor in this connection. Whereas nothing but a thickened dura may be found in a timely operation, delay would in all probability have led to the exposure of an ulcerated membrane. The findings are moreover to a certain degree dependent upon the species and virulence of the invading microorganisms.

Extradural abscess, provided it is exposed in time, with thorough removal of the pus, is very amenable to treatment.

Intrameningeal, or Intradural Abscess (pachymeningitis interna) occurs as a circumscribed lesion, without general infection of the subdural spaces, and is the result of disintegration of the dura, with involvement of the subdural spaces by the pathologic process. A certain amount of infectious material the dura is able to cope with,—in a way similar to the peritoneum,—without giving rise to a generalized infection The intradural spaces are protected by the short duration of the local process, and the diminished virulence of the infection.

Koerner (Die eitrigen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter) describes two types of intradural abscess: (1) Subdural abscess, accompanied by ulcerations on the brain cortex. (2) The formation of intradural abscess, with a somewhat diffuse involvement of the intradural spaces. The writer interprets these conditions as differences of degree rather than of kind, in the sense outlined above. Cases of the first class may remain latent for a certain length of time, without giving rise to symptoms of any sort (Observation 6). As a rule, the middle cranial fossa is involved.

Surgical interference is followed by prompt relief, as illustrated by Case 6. The presence of a similar abscess can hardly be recognized prior to the operation.

The second group of cases is characterized by the simultaneous existence of extradural abscess, sinus thrombosis, or actual brain abscess (Observation I).

The clinical picture is dependent upon the distribution of the lesion and its manifold complications. In the vast majority of the cases, the diagnosis is not rendered until the patient comes to operation. A recognition at this time of the true state of affairs, with emptying of the pus, means a decided and speedy change for the better. The patient's prospects are less favorable in the diffuse conditions, where the inflammation reaches for into the intradural spaces (Observations 1 and 10).

Certain mixed cases occur, which present the features of both extradural and intradural abscess, sometimes in combination with rather deep ulceration or necrotic areas of the brain surface proper (Observation 6). These cases represent a transition form between extradural and intradural disease, and cannot be correctly included under either heading.

Purulent Otitic Meningitis occurs as an involvement of the pia mater, and may appear as an independent but simultaneous process, together with sinus thrombosis or brain abscess. Again, it may be the sequel to any of these lesions. It was observed as the terminal lesion in cases 1, 2, 3 and 4. It is a relatively uncommon, remote result of middle ear suppuration in its uncomplicated form; brain abscess or sinus thrombosis being the more usual sequela. Sometimes superficial brain ulcerations are noted Martin (quoted by Huguenin, Ziemssen's Handbuch, Vol. IV), Bürkner (Arch. f. Ohrenheilk., 19, Case 3), Bezold (Arch. f. Ohrenheilk., 21, Case 5) and other observers reported small surface abscesses. The ventricles may be invaded by the pus, as shown in a personal observation.

The point where the meninges are in contact with the diseased bone is the favorite site of the greatest pus accumulation, which is accordingly looked for at this demonstrable point of contact. (Held and Koptezky, Arch. of Otology, Vol. XXXV. The case upon which this observation was based, is republished in this communication as Case 7). This area constitutes the starting point of the meningeal involvement.

In those cases where a labyrinthine infection is responsible for the intrameningeal purulency, the meninges are generally found to have become primarily affected in the cerebellar region, as illustrated by observations 2, 3 and 4.

Although the exposure of the dura in the cerebellar region, in Cases 3 and 4, plainly showed extreme intracranial tension, the lumbar puncture did not demonstrate cerebrospinal fluid under pressure in the spinal canal. A considerable quantity of purulent fluid escaped from the arachnoid space, when the dura was incised, after negative pressure had been found at the lumbar puncture.

The discussion of the diagnosis of purulent meningitis does not lie within the scope of this article. The same remark applies to a detailed consideration of cerebral and cerebellar abscess.

Brain Abscess is usually produced by a chronic middle ear suppuration of prolonged standing (a number of years: three in Case 1; eighteen, in Case 2). The majority of brain abscesses are the result of actual disease of the bone, rather than lesions of the mucous lining of the middle ear cavities. For this reason, especial importance attaches to the differential diagnosis between a chronic middle ear suppuration associated with bone disease, and one in which the mucosa alone is affected. Although necessarily delicate, this differentiation is nevertheless possible to a great extent by the study of the otoscopic picture, combined with bacteriologic and cytologic examination of the discharges from the ear.

Adhesions between the dura and the surface of the brain are frequently observed in brain abscess, which should accordingly be suspected whenever such a condition is found to exist.

TREATMENT.

The treatment of these intracranial lesions is governed by the local findings in individual cases. Each patient will have to be treated according to individual requirements, and under these circumstances, it is not possible to map out a general plan of treatment adapted to all cases. The management of these lesions is based upon the following principles:

I. The radical extirpation of the primary pathologic foci in the temporal bone, by means of a careful surgical exploration of the bony wound cavity (deep general anesthesia being usually required), for the uncovering and removing of neglected purulent mastoid cells; with examination of the exposed sinus wall and the tegmen dura. A wider opening of the middle ear spaces is indicated in cases wth a suggestion of labyrinthine involvement; here the wall of the labyrinthine capsule must be explored for evidence of the route of invasion of the internal ear. After the infection of the labyrinth has been demonstrated, a further exploration of the internal ear is required in the presence of defects in the walls of the semicircular canals, or ulcerations of the labyrinthine windows, or perforations in the cochlear convolutions. The internal ear is to be explored upon the basis of positive findings only, since this interference represents a very grave surgical procedure, which moreover implies the destruction of hearing in the affected ear.

II. The evacuation of extradural, intradural and brain abscesses, with establishment of meningeal drainage. The condition of the exposed sinus wall is revealed by its appearance, coloration, touch, etc. A perisinus abscess is emptied by the mere exposure of the sinus wall. The uncovering of a small area of dura over the tegmen is harmless in itself, provided the dura is healthy. Its systematic peformance in doubtful cases will frequently serve to reveal the presence of unsuspected extradural gatherings of pus. Intradural lesions may be suspected upon the evidence of the exposed dura (aspect, color, feel) in combination with existing intracranial tension. This suspicion is confirmed when the dura is found to be covered with discolored granulations, pus escaping between the meshes.

Large areas of necrosis in the dura are removed, while small necrotic spots are allowed to remain. An incision of the dura provides for the escape of intradural pus. Large collections are best managed by means of exposure through the squamosal plate, and application of a counter-opening, for the facilitation of meningeal drainage. Multiple parallel incisions in the dura have been found serviceable. The evacuation of an abscess in the sphenoidal lobe, or puncture of the lateral ventricle, if required, is easier of performance from the opening in the squamosal plate, than by way of the existing wound.

Exploration of the sinus, and eventual ligature of the internal jugular vein, may become necessary by the devolpment of sinus thrombosis, as a sequel to the evacuation of a perisinus abscess. Under these conditions, surgical interference is indicated at once after the diagnosis has been rendered. A cerebellar gathering of pus is suggested by a continual high temperature, with a healthy wound, after ligature of the internal jugular vein, and evacuation of the clot, in sinus thrombosis. This indicates the removal of the inner mastoid table overlying the cerebellar meninges. After the cerebellar abscess has been located, the pus may be reached either by way of the mastoid wound, or through the cortex, posteriorly; the procedure to be adopted depending upon the location of the abscess in a given case.

III. The maintenance of normal intracranial pressure, as far as feasible, through repeated lumbar punctures. The diminution of intracranial pressure, through the withdrawal of cerebro-spinal fluid, is of importance, apart from any diagnostive data this may furnish. Relief and amelioration were invariably shown in the writer's experience, after lumbar puncture, by a clearing of the consciousness, a diminished rigidity of the back, and as a rule an improvement of the patient's general condition. The progressively diminishing purulency of the puncture fluids obtained from the favorable cases, at different dates, is of auspicious augury and stimulates further efforts in the interests of the patient. Meanwhile, such an outcome is reserved for the cases with a patent foramen of Magendie. The prognosis is a gloomy one when this outlet is obstructed, with a negative pressure of fluid in the spinal canal, and high tension of fluid in the subarachnoid space intracranially.

RESULTS OF OPERATIVE INTERFERENCE.

Among the ten cases, there were four recoveries and six deaths. The fatal outcome was due in one case to septic pyemia, the operation coming too late to prevent the effects of the profound general sepsis. In a second patient, a child, death occurred presumably from shock, before the operative results could be determined. In the remaining fatal cases, the surgical intervention failed to arrest the progress of the disease.

The fatal outcome is not necessarily governed by the wide distribution of the pathologic process. Recovery followed in one case of the series (Observation 7), although there was extradural and intradural pus, with involvement of the lateral ventricle and spinal cord. A fatal ending was noted in those cases in which the posterior cranial fossa was the seat of the main foci of the lesion. The bone disease could be traced directly to the meninges by contiguity in only one of the fatal cases (Observation 10). The invasion of the posterior cranial fossa was shown beyond a doubt to take place by way of the labyrinthine channels, as illustrated by the gross operative findings in Cases 2, 3 and 4. Moreover, this was borne out by the clinical picture. (Autopsies were not permitted.) It is the writer's impression that the outcome in some of these cases would have been a different one, if the disease had been arrested in the labyrinth.

The meningitis and abscess formation in the cases terminating in recovery (Observations 6, 7, 8 and 9) were caused by direct contact with diseased bone, without involvement of the labyrinth.

Judging from current experience with these lesions, it appears that meningeal inflammation by contiguity offers more favorable chances for a successful outcome after operation, than does meningitis subsequent to invasion of the labyrinth. The middle cranial fossa is more tolerant of the purulency and responds more promptly to its surgical evacuation, than the posterior cranial fossa. Gatherings in the last named location are not permanently relieved by surgical interferences, without preliminary control of the purulent process in the laybrinth.

In conclusion, I urge that expectant treatment in the face of post-mastoidal intracranial symptoms be limited as much as possible, and that as soon as diagnosed, or even a well-founded suspicion of purulent involvement of the meninges is entertained, that a systematic exploration of the meninges and labyrinthine capsule be undertaken.

It is not advisable to delay the operation on the labyrinth until the appearance of the meningeal symptoms. These will obscure the symptoms pointing to the labyrinth, and thus serve to diminish the patient's chances for ultimate recovery. A number of cases which would go on to general meningitis and death will be saved by the early establishment of meningeal drainage.

Exploratory operations are indicated in even the most desperate cases, from the clinical point of view. Remarkable results have been known to follow, and precisely among the patients otherwise doomed to death, one or the other may have the scales turned in his favor, contrary to all expectations.

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III.

OCULAR SYMPTOMS OF INTRACRANIAL COMPLICATIONS IN OTITIC DISEASE.

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The progress of otology during the past ten years has been so largely practical and surgical that even the operations for intracranial complications have lost much of their originally formidable, not to say ominous, aspect. The otologist of today is, theoretically and technically, prepared, in case of definite indications, to follow up the suppuration from its source in the middle ear or mastoid whithersoever it may lead him. It is now generally recognized that complicated methods of localization and the use of base lines, measurements and cranial topographic studies are of minor importance, as we have learned that the intracranial pus accumulations are, in a majority of cases, to be found in the immediate neighborhood of the purulent infectious focus in the temporal bone, or at least that they can most easily be reached, treated and drained by an extension of the original field of operation. While this increase of security in operating and the consequent improvement in surgical statistics render the question of diagnosis less immediately vital and important, they by no means lessen its scientific import or make it any less incumbent to place upon a definite basis, as far as possible, the question of the significance of intraocular changes in the diagnosis and prognosis of endocranial complications of purulent otitis.

An interesting feature in the study of ocular symptoms in otitic intracranial complications and one of importance in the history of investigations on this subject is the fact that in this country many ophthalmologists are aural surgeons as well. A generation ago, when the surgery of otitic disease was in its infancy, this was almost invariably the case, and it is due in large measure to the work of Kipp, Gruening, Knapp, Roosa, Alt and other Americans that the teachings of Politzer, Bezold, Zaufal, Lucae, Troeltsch and Schwartze have been spread in this country. Kipp was, in fact, the very first to call attention to the importance of making ophthalmoscopic examinations in cases of ear disease. His first article[1] on this subject antedated that of Zaufal by a year, yet the latter is always spoken of, at least by German authors, as the pioneer in this field. Kipp's second article[2] appeared in 1885, and the conclusions as to the value of ophthalmoscopic changes in prognosis and treatment stated in his second paper are now generally accepted. Hansen urges the importance of the otologist being skilled in ophthalmoscopy in order to insure early and repeated examination of each and every case. American otology recognized and fulfilled this requirement years before it was formulated by Hansen in Germany.

Diagnostic signs are of practical import, whatever their scientific and theoretic value may be, as far as they serve as a basis for therapeutic considerations, prognosis or operation. This does not imply that a study of symptoms in cases of diseases which for other reasons are beyond the reach of therapeutics, at the time, is lacking in academic interest or in eventual practical importance, as the progress of methods of treatment may finally come abreast of the diagnostic methods. In the sphere of brain surgery or that part of it associated with the intracranial complications of otitic disease, the practical indications to explore the cranial cavity and determine the condition of sinuses, meninges and central organs are quite definite and generally urgent. The making of an accurate diagnosis hardly aids in treatment in this field. On the other hand, there is a development of other diagnostic methods, among which we may mention lumbar puncture, the blood examination and differential leucocyte count, the careful examination of the reflexes, Kernig's and Babinski's signs, the careful bacteriologic examination of aural discharge and of tissues removed from mastoid, sinus or jugular vein at the time of operation.

Inflammatory changes or circulatory disturbances in the optic nerve have always been considered corroborative evidence of similar endocranial conditions causing an increase of brain pressure or indicating inflammatory processes. In the otitic complications the evidence is often contradictory, obscure or insufficient. The views of the most experienced observers and the opinions expressed by standard authors on this point do not agree perfectly.

Bacon.[3]—Temporosphenoidal abscess. Optical aphasia a symptom of the highest diagnostic value. The patient, when shown an object, knows it, but is unable to name it (better, has no conception of its name; the other phrase might mean that he could not speak the name he had in mind, i. e., motor aphasia), but he is able to recognize and understand its name, and will often describe the object. He can sometimes name the object if allowed to handle or smell it (showing there is no motor aphasia, and that a conception of name, due to stimulation of memory pictures, can be accomplished over other paths than the visual). In one case there was, besides the difficulty in naming an object, difficulty in calling to mind its appearance, visual amnesia (Starr), "due to a suspension of function in association tracts lying deep in white matter of temporo-occipital lobes."

Inflammation of optic nerve may or may not be present. More frequent in cerebellar abscess. Symptoms of latter may be simulated by disease of semicircular canals. In the case of a child with suspected cerebellar abscess characteristic symptoms, including choked disc with retinitis, were entirely abolished by removal of granulations from the middle ears and establishment of proper drainage. In an abscess of the right cerebellar hemisphere there was edema of the right optic nerve. Photophobia and nystagmus may occur.

Sinus Thrombosis.—When the clot has extended to cavernous sinus, and ophthalmic vein has become engorged, there is likely to be a certain amount of edema of the eyelids on the corresponding side Neuroretinitis is noticed in some cases, more especially in the later stages, but it is not constant.

Purulent leptomeningitis. Photophobia, pupils contracted and responding but little to light, and optic neuritis. Later the pupils are dilated and often unequal. In children, symptoms suggestive of meningitis may be due to retention of pus in the middle ear. When paralyses occur, and there are optic neuritis and other characteristic symptoms, the diagnosis is assured. (Koerner does not lay much stress on neuritis in meningeal irritation in children, and cites a case in which he was urged to operate on account of double choked disc. It was a serous meningitis of mild type, and, as pulse and general condition were good and weight was increasing, he temporized. Recovery was spontaneous.)

Blau.[4]—It is advisable to consider any disturbance of ocular motility occurring in the course of an otitis media as suggestive evidence, symptom of secondary involvement of the labyrinth or of intracranial complications. For the diagnostic value (Verwerthung) of ocular symptoms in the diagnosis of intracranial complications, it is of prime importance to determine whether morbid changes, particularly of the fundus, can be induced by middle ear disease. Cases cited to substantiate such a possibility were all suspicious of already existing complications, and were so considered by the observers themselves. Ocular symptoms are, then, of definite significance, to the extent that they appear to develop only when otitis has involved the interior of the skull, with the exception of nystagmus, which may be excited as a reflex on irritation of the "tonus labyrinth" (semicircular canals). Hyperemia of the nerve, neuritis, choked disc, render it highly probable that we have to deal with threatening or existing intracranial complication, but do not justify any conclusions as to their nature. On the other hand, the absence of intraocular changes by no means excludes the presence of intracranial suppuration, so that in the presence of other characteristic symptoms the negative result of ophthalmoscope has absolutely no significance. It is of importance as corroborating the other symptoms and clinching the diagnosis when present.

Burnett[5] quotes T. R. Pooley to the effect that "if the optic neuritis is found in connection with purulent inflammation of the ear, the diagnosis of extension of ear disease to the brain is certain, no matter whether other evidence exists or not." Unfortunately it does not explain the nature of the intracranial lesion, and, while optic neuritis in chronic otorrhea, or even acute otitis, would be an urgent indication for immediate mastoid opening, its occurrence must be considered only in connection with other symptoms as an indication for an exploratory opening into the cranium.

Optic neuritis is usually not observed, or only slightly marked, in uncomplicated sinus thrombosis; it is sometimes found with extradural abscess. In cavernous sinus thrombosis, due to the passage of infection from the middle ear by the carotid canal (Koerner) edema about brow and orbit, exophthalmos, paralysis of extraocular muscles, immobility of the globe (due to orbital cellulitis), edema of lids, chemosis of conjunctiva and choked disc on the side of the chronic aural suppuration, are seen.

In otitic cerebral abscess and in extradural abscess neuritis is frequent. Optic acoustic aphasia is usual in abscess of left temporal lobe, or in the right lobe if the patient be left handed. Ptosis of left upper lid has been seen in left temporal lobe abscess (Steinbruegge). Sensory aphasia with homonymous hemianopsia with preservation of pupillary reaction points to temporal lobe.

Symptoms of extradural abscess or of sinus thrombosis always dominate and mask those of brain abscess. This applies to fundus changes also.

Cerebellar abscess shows varying pupils, the wider being on the side of the abscess, conjugate deviation away from affected side, horizontal nystagmus, abducens paralysis, lagophthalmus, due to facial paralysis. Double optic neuritis is often the only symptom, but the fundus may remain normal even in fatal cases[6] Cerebellar abscess is very likely to obstruct circulation of cavernous sinus and ophthalmic vein.

Meningitis.—No mention of ocular symptoms.

Bezold.[7]—Labyrinthine suppuration shows, at least in early stages, characteristic symptoms of irritation, subjective dizziness, vertigo even while lying down, disturbance of gait, horizontal nystagmus when gaze is directed to the sound side, and vomiting. Nystagmus may be rotary and always disappears (destruction of ampullar organs) sooner than the dizziness. Often complicated by fatal meningitis and cerebellar abscess.

Extradural pus collections may give no clinical signs.

Operate after eight weeks in acute otitis, and after several months of fruitless treatment of fetid discharge in chronic cases to find the source of continued suppuration.

Choked disc and double edema of both lids speak for thrombosis of both cavernous sinuses.

In brain abscess, optic neuritis, oculomotor or abducens paralysis. Otitic leptomeningitis may start from any point of a sinus thrombosis, or from brain abscess which has ruptured into ventricle or subarachnoid space. Extradural abscess may cause necrosis of dura and spread to arachnoid and pia.

Dench. Serous meningitis (1903).—Most frequent paralyses those of the third, fourth and sixth nerves. Paralysis of the third nerve causes at first contraction and later dilatation of the pupil. An early symptom is failure to respond to light. Where local paresis exists, divergent strabismus is probably the most frequent symptom. The fundus will most generally show either a choked disc or an incipient swelling of the optic papilla. It is strong corroboration of the other symptoms of meningitis, but is found in any intracranial lesion which increases pressure. Sinus thrombosis. Presence of choked disc will confirm the diagnosis in doubtful cases.

Brain abscess.—Choked disc may be present, but is not characteristic of abscess alone.

Cerebellar abscess.—Choked disc is often present.

Brieger.[8]—Fundus changes are generally missing in uncomplicated extradural abscess, and arouse suspicion of other endocranial complications. Extradural compression causes disturbances in fundus which are rapidly compensated. Repeated examination may detect transitory fundus changes. Abducens paralysis is not infrequent. Kessel reports paralysis of abducens and oculomotor nerve of the opposite side, due to collateral edema of meninges or brain, and not to direct compression.

Purulent leptomeningitis. While Pitt found the fundus normal in all uncomplicated cases and while Knies claims optic neuritis as the most valuable diagnostic aid, the truth lies in the middle. His experience agrees with that of Hansen, that about half the cases show changes of the fundus which are sufficiently marked and unambiguous to allow a diagnosis. But these changes never appear as typical choked disc. They are rather in the nature of optic neuritis, and vary from slight hyperemia to entire obscuration of the margin of the disc. Generally double, occasionally unilateral, or unequal in degree, or even more marked on the side of the sound ear. In the cases with insidious onset optic nerve changes were rarely seen at an early stage before manifest symptoms developed. The pupils show a markedly rapid variation between contraction and maximum dilatation.

Abducens and oculomotor paralysis are not always due to localization of the purulent process at the base, but may be due in still circumscribed meningitis to general increase of pressure in the cerebrospinal fluid and be present as the only symptom for some time before the meningitis becomes generalized.

Nystagmus may be present in the form characteristic of labyrinthine disease or, more correctly, of implication of the semicircular canals, i. e., horizontal oscillations when the gaze is directed toward the sound side. After generalization of the purulent process, and not infrequently after operation, the nystagmus also becomes generalized in the sense of occurring with all ocular rotations, either constantly, and even when the eyes are at rest or the lids closed, or only when an object is fixed. This would indicate a spreading of the suppuration from the horizontal to the other two semicircular canals, independent of movements of the eye.

Conjugate deviation is rare.

Serous meningitis. Eye symptoms, as neuritis of slight degree, never as choked disc, with only slight hyperemia of the papilla. May be transitory or develop early, but may also appear later (Hegner) even several days after increase of intracranial pressure had been relieved by operation. Abducens paralysis may be the only symptom of serous meningitis locally, due to compression at the base. A case of nuclear lesion on the floor of the fourth ventricle, causing paralysis of associated motion (Blick-laehmung) has been reported by Brieger.

Kirchner.[9]—Cavernous sinus thrombosis may develop early not only in the chronic but in acute otitis media, especially after influenza. Exophthalmus, edema of the lids, paralysis of extraocular muscles, disturbance of vision, are the usual symptoms.

Kyle, J. J.[10]—Purulent leptomeningitis. Contraction of pupils, loss of light reflex, later dilatation.

Thrombosis. Optic neuritis is one of the symptoms (quoted from Ballance). Extradural abscess. Occasionally optic neuritis a suggestive symptom.

Love, J. K.[11]—Meningitis. Pupils contracted, reaction to light lessened. Later, optic neuritis is often present, pupils unequal and dilated. Ptosis and squinting may occur.

Sinus thrombosis may simulate meningitis. Optic neuritis may be present. Brain abscess. In early, irritation stage, pupils contracted, photophobia. Later unequal and do not respond to light. In temporosphenoidal abscess, third nerve may be involved, and ptosis, accommodation, paralysis and oculomotor paralysis of all but external rectus (abducens) occur. Optic neuritis occurs in about half the cases of cerebral and 60 per cent. of cerebellar abscess.

Koerner.[12]—Extradural abscess. Neuritis not unusual (Pitt, Lane, Johnson, Zaufal). Of 11 uncomplicated cases, 8 normal. Of 20 uncomplicated in Schwartze's clinic (Grunert), all normal. Koerner cites two cases, however, of marked changes, both in children, with extradural abscess in posterior fossa. In one case the neuritis increased after evacuation of the abscess, particularly on the sound side. In the other, double choked disc, also more marked on the sound side, first appeared after operation. Cure in both. There was a possible complication with sinus affection in both.

Knies' view as to the importance of optic neuritis in diagnosis of meningitis is not shared by Koerner. The cases of Kipp and of Andrews were not subjected to autopsy, and the histories read like complication with sinus phlebitis and extradural abscess, respectively. In serous meningitis, particularly in children, optic neuritis has been observed in light as well as in severe cases. This is the more surprising, as neuritis is missed so often in purulent meningitis.

Thrombosis, neuritis and choked disc common in cavernous sinus thrombosis. Koerner has not seen it in sigmoid sinus thrombosis, uncomplicated. Leutert saw neuritis in 1 of 10 cases, Hansen, 3 in 8 uncomplicated cases. Jansen finds the changes often and finds them of diagnostic value.

Jansen has seen nystagmus in sinus thrombosis and extradural abscess.

Brain abscess. Neuritis frequent, but not invariable. Generally double and more marked on the side of the abscess. Late symptom and not marked, and may advance after evacuation of the abscess. Sight often but slightly affected, and consecutive atrophy rare Hansen, 12 cerebral abscess, 2 with vessel changes and 4 with optic neuritis; 7 cerebellar, 2 optic neuritis. In one of the latter, neuritis only on side of abscess, in 3 double and equal, in 4 more marked on abscess side.

Total amaurosis seen in 3 cases by MacEwen and Berridge, reported in detail by Koch (Otit. kleinhirn Abscess., Berl., 1897). Operation in one case, with disappearance of amaurosis. In one fatal case there was complicating ventricular serous meningitis and purulent leptomeningitis. Case 3, autopsy incomplete. Nothing similar has been observed in cerebral abscess.

Hemianopsia generally due to lesion of the internal capsule, or when situated in occipital lobe to subcortical lesion of the optic radiation. These are distant symptoms due to pressure of abscess on neighboring regions and edema of brain substance.

Oculomotor palsy frequent. Temporosphenoidal abscess of at all considerable size invariably causes paralysis of some side, generally incomplete and limited to pupillary fibers and levator (ptosis and accommodation paralysis), occasionally involving all branches.

Pupillary mydriasis common in cerebellar abscess.

Abducens paralysis may be homolateral or crossed. It is of little diagnostic value, occuring in serous meningitis and spontaneously healing complications.

Conjugate deviation may be toward side of abscess. (Kalmus, Moos) or away from it (Schwartze, Acland and Ballance, Hansberg, Winter and Deansley). Nystagmus (Mueller, Winter and Deansley, Ballance, Barling, Okada). It is a question whether this is not a labyrinthine symptom.

Partial or complete oculomotor paralysis is a valuable sign of temporosphenoidal abscess.

In children, brain tumor (tubercle) not infrequently complicates suppuration in the temporal bone. Choked disc speaks more for tumor, neuritis for abscess. Chorioidal tubercle may be discovered.

Knapp, H.[13]—Pachymeningitis externa; epidural or extradural abscess. Symptoms of meningitis are present in irritation in many cases—headache, slight rise of temperature, choked optic disc, pressure pain, somnolence, slowing of pulse, stiffness of neck, but these symptoms are too indefinite to make a diagnosis.

Pachymeningitis interna, subdural or intradural abscess with softening and abscess of adjacent brain substance—circumscribed encephalitis—occurs when pia and arachnoid are agglutinated over inner surface of an infected area of the dura and exudation or perforation takes place. If not adherent purulent leptomeningitis results. Optic neuritis is rare.

Sinus thrombosis and pyemia. As the last of eight symptoms in importance and frequency, Koerner mentions stupor and optic neuritis as present only in complication with encephalic abscess and meningitis.

Brain abscess, four stages, initial, manifest, latent, terminal. Optic neuritis, earlier and more pronounced on the diseased side, is one of the symptoms of brain pressure.

Homonymous hemianopsia, on record seven times, probably would have been found oftener if looked for regularly.

Partial or total paralysis of the motor communis oculi and abducens nerves, conjugated deviation of the eyes (to either side), and nystagmus, are rare and indefinite symptoms.

Lake, R.[14]—Meningitis. In progressive cases, intolerance of light, at times amounting to photophobia, becomes marked. The pupils, which are commonly unequal, react to light, but usually more or less slowly than normal. Optic neuritis or papillitis will in most cases be noted. After development of the lethargic state the pupils often become widely though unequally dilated, and all reaction to light is gradually lost, while in basic cases a tendency to squinting is more frequently noted. Nystagmus may be present in later stages.

Extradural abscess. It is rare to find optic papillitis in uncomplicated cases.

Brain abscess. Pupil on affected side frequently contracted and only reacts sluggishly to light and accommodation, in which case the abscess will be found to be small, relatively, but if it is of large size the pupil on the same side will be both dilated and stable. Paralysis of the third nerve on the same side as the abscess is a variable sign; optic neuritis, or papillitis, is found during the whole of this stage and may be only on the affected side.

Cerebellar abscess. In second stage there is not infrequently conjugate deviation of the eyes to the side opposite to the lesion, along with optic neuritis. Lateral nystagmus with large excursions is especially frequent in lateral lobe abscess, the movements of the eyeball are toward the side on which the abscess is situated. Rotary nystagmus points toward involvement of the middle lobe. (These considerations seem to be largely hypothetical. If the movements of the eyeball are toward the side, any side, there must be deviation, if not, there is merely horizontal nystagmus. Lake may mean that nystagmus develops only when the eye looks toward the affected side.—P. H. F.)

A final decision on such points is often impossible until a large number of cases has been collected, critically studied, classified and analyzed and a statistical study made of the results. Statistical studies of this sort have been made in Germany by Sessos, Hansen, and by Koerner, whose cases were supplemented a year or two later by Takabatake. In this country there have been no analyses of large series of cases, although various otologists have reported their observations of specially interesting, grave or successfully treated cases of brain abscess, meningitis and sinus thrombosis.

Hansen's statistics comprise 97 cases, of which 45, about 46.4 per cent., showed optic nerve changes, while Koerner reports 54 cases with positive ophthalmoscopic findings in 16, or 29.6

per cent. Sessous (Lucae Festschrift, 1905) discusses 104 cases, 5 per cent, of all those treated at the Berlin clinic from 1895 to 1905. Ophthalmoscopic changes were most frequently noted in meningitis complicated with sinus thrombosis, least often in extradural abscess. A normal fundus is no proof of absence of intracranial complication, nor does the presence of fundus changes allow us to draw any but the most limited and cautious conclusions as to their presence. This also holds true in regard to prognosis as to recovery. Fundus changes are of diagnostic importance only when other cerebral symptoms are lacking. This is in marked contrast to the view of Ostmann cited above, that fundus changes clinch a diagnosis already made probable by other symptoms, but are of no value by themselves, and to the opinion of Koerner and of Hansen, who attach great importance to positive findings in the ocular fundus in suspected cases of intracranial suppuration. Hansen sums up his observations as follows: "Fundus changes may occur in any intracranial complication. Least frequently (18 per cent.) in extradural abscess, and then more often in the deep seated than in the perisinuous variety, they are most often observed, 87.5 per cent., in cases which had been considered as hyperemia of the brain and meninges, or serous meningitis, in 57 per cent. of uncomplicated, and 47 per cent. of complicated purulent meningitis (about 50 per cent. of all meningitis), in 50 per cent. of cerebral, and 29 per cent, of cerebellar abscess, 37.5 per cent. of uncomplicated and 47.5 per cent. of complicated sinus thrombosis (agreeing with Jansen). In other words, fundus changes are about equally frequent in the various forms of otogenous intracranial suppuration, occurring in about 44 per cent. Neuritis is the most common condition, slight neuritic change, vessel changes on the disc next in frequency, while real choked disc is unusually rare (3 per cent.) and occurs only under certain definite conditions. Both eyes were affected in the great majority (80 per cent.) of positive cases, and the changes were usually (75 per cent.) more marked on the affected side, although the opposite condition was noted in 9 per cent. Unilateral involvement of the fundus, observed in 20 per cent. of the positive cases, was invariably situated on the side of the ear affection. In brain abscess the fundus changes were almost invariably (87.5 per cent.) bilateral, and more marked on the affected side (50 per cent,) or of equal intensity (37.5 per cent.). Hansen does not agree with Koerner, MacEwen and

Oppenheim that fundus changes in brain abscess are not infrequently found on the side opposite the intracranial lesion. As other symptoms of intracranial complications are often variable, evanescent, scanty and obscure, and the recognition of these ominous conditions consequently difficult and uncertain, positive evidence of fundus changes, which can always be detected when present (except in cases of opacity of the media, as cataract, etc.) is of undeniable and decided value. Their development in otitis suppuration always indicates the presence of some intracranial complication, for it is an unproven and improbable assumption that middle ear disease alone, without endocranial suppuration, can induce morbid changes in the optic disc. As such changes are found in only about one-half of all cases of intracranial complication, their absence is never to be considered as positive evidence. Fundus changes are of definite importance in suppurative otitis, as their development, even in the absence of all other cerebral symptoms, indicates some intracranial complication, and accordingly a more severe affection. The prognosis of intracranial complication itself, on the other hand, is not influenced by the presence of fundus changes, as the latter may occur in the least dangerous as well in the most serious forms, and in mild as well as in severe or even fatal cases of a single form. Ophthalmoscopic examination should be made in every case of otitis suppuration, and repeated frequently in case there is any unusual feature in the progress of the case or even the slightest cerebral disturbance. The otologist should carry out these examinations himself, as this is the only guarantee for their regular methodical performance and record. Disc changes are an immediate indication for opening the mastoid in a case of middle ear suppuration which for any reasons has not been operated. The subsequent course of the case must then decide whether further surgical intervention is necessary.

Koerner agrees in the main with Hansen as to fundus changes being bilateral, even when the intracranial disease is limited to one side, in the great majority of cases, and as to the presence or absence of fundus changes having no significance for the prognosis of the intracranial complication. In purulent meningitis he comes to quite different conclusions, all of his 6 uncomplicated cases having had normal optic disc up to the time of death, while of the 8 complicated cases 6 showed fundus changes. Simple thrombosis caused disc changes in only 1 of 5 cases. One-sided occlusion of the cranial sinuses, it seems, is not sufficient to produce venous congestion in the eyeground, and this applies as well to the sudden disturbance of circulation incident to jugular ligation as to the more gradual stoppage by thrombosis or endophlebitis. Only about 25 per cent. of all cases of intracranial suppuration showed optic nerve changes. They are more generally absent than present, and more apt to indicate a combination of conditions than any one complication. Thus of 18 cases of a single complication only 3 showed fundus changes, and even in these there was some suspicion of another form of intracranial suppuration. Koerner sums up as follows: The variety of fundus change. neuritis and choked disc, respectively, does not allow any conclusions as to the character of the intracranial complication. A more marked development of fundus changes on one side does not indicate exclusive or even predominant involvement of the corresponding half of the cranium. Fundus changes do not influence the prognosis, and even advance of these changes after evacuation of pus in the cranial cavity does not alone make the outlook any worse.

My statistics and conclusions are based on a study of the records of the New York Eye and Ear Infirmary from 1905 to 1907. Of a total of 19,129 cases of ear disease treated during these two years, 11,176 were affections of the middle ear and Eustachian tube, 1,064 affections of the mastoid. There were 11 cases of cerebral, 25 of epidural, and 73 of subperiosteal abscess; 12 of jugular vein and 13 of lateral sinus thrombosis. These figures represent the cases so diagnosticated on admission. The full number of complicating intracranial cases is to be determined only from the report of operations which is shown on Table 1, which shows 16 exploratory and 65 curative operations for sinus thrombosis, 24 exploratory and 6 curative for brain abscess; 31 ligations of the internal, and 1 of the external jugular, and 2 extirpations of the internal ear for labyrinthine suppuration. These cases were operated on by or in the service of Drs. Dench, McKernon, Bacon, Whiting, Lewis, Adams, W. S. Bryant, Rae, J. D. Richards, Stevens, Kenefick and Brandegee.[15]

A study of these records shows that the great majority of cases of intracranial otitic complications were examined at some time or another during the stay in the institution, for evidences of changes in the fundus oculi. A few cases were not so examined, either on account of death having come on suddenly a short time after admission, or because the classic symptoms of intracranial disease assured the diagnosis, or finally because the symptoms receded immediately ofter operation. These reasons are of practical weight, but from a scientific point of view it is to be regretted that systematic examinations of the fundus are not made in every case of operative ear disease. The presence of choked disc or of an optic neuritis several days after operation is open to several interpretations in regard to the period of development of the causal complication, whereas its significance is very much less ambiguous in case a normal fundus was found at a previous examination. Koerner lays stress, and, justly, on the importance of repeated examinations "up to the time of cure or death, i. e , until the diagnosis has been established, etc." The corroboration of the ophthalmoscopic findings, or rather of the diagnosis which they are to assist, is unfortunately beset with more than usual difficulty. In the case of recovery the character of the ailment is often no more positively determined than it was before, or, at all events, there is no certainty of such degree as to enable us to stamp the diagnosis as agreeing with the intraocular changes or not. The findings at operation may be of value, especially in the event of positive evidence, in the form of the evacuation of pus, of a brain abscess, of an occluded, thickened or ulcerated sinus, discolored meninges or subdural abscess formation, but in other cases they fail to clinch the diagnosis. The only absolute verification, namely, by examination postmortem, is excluded, of course, in case of recovery, and frequently by the inability to obtain permission for autopsy. This is the rule rather than the exception, and unfortunately it is just in the most serious cases, those dying of cerebral complications, that an addition to our definite knowledge would be of greatest value. From this point of view our statistics, no matter how large, are much less instructive than the smaller ones of continental, and particularly of German, observers, who have been enabled in the great majority of cases to corroborate or disprove the intra vitam clinical diagnosis by autopsy and pathologic examination postmortem. The postulate of examination by experienced and skilful ophthalmologists is fully met in the case of the institution cited in my study, as it has a large and active eye department, and numbers among its surgeons Gruening, Bull, Derby,[16] Callan, Weeks, Marple and other well-known oculists. The internes, too, have with few exceptions had a double service, with ample practice in the use of the ophthalmoscope and the study of fundus conditions both in the in- and out-patient department. In many instances the name of the examiner is mentioned, and in some, several consultants have expressed their opinions as to the fundus conditions. Where no examiner is specified the ophthalmoscopic findings were made by the house surgeon. In case of doubt or of unusual conditions the attending surgeons were called on for an opinion. As in many other reports of fundus conditions accompanying otitic complications, it appears that there is not sufficient attention paid to the necessity of a strict differentiation between optic neuritis and choked disc, or, in the case of already established atrophy, to the distinction between the simple and the postneuritic form. While in the latter case it may not always be easy even for the experienced ophthalmologist to detect slight traces of previous inflammation about an atrophic disc, such as irregularity of outline, proliferation or irregular distribution of pigment along the disc margin, filling up of the lamina cribrosa with connective tissue, irregularity of lumen, thickening of vessel walls or connective tissue striæ in their course, some or most of which changes may be wanting, there is usually no doubt at all, in a specific concrete case. whether we have to deal with neuritis or choked disc, at least in the early stage. And here, again, is a reason for early as well as for repeated ophthalmoscopic examinations; a choked disc may be engrafted or superadded to a neuritis, or *vice versa,* in a case, respectively, of an intracranial inflammation, such as meningitis being complicated by a process which greatly increases brain pressure, as, for example, brain abscess or internal hydrocephalus, or, on the other hand, in the still more unusual event of inflammatory changes in the meninges occurring in brain abscess or thrombosis.

The scope of this presentation does not permit of a detailed consideration of the pathogenesis and mechanical development of choked disc and optic neuritis, a subject of practical import for the interpretation of clinical pictures in cerebral disease, particularly as there is still a difference of opinion as to the exact manner in which these intraocular conditions are brought about, and a number of conflicting theories to explain them. We may in a general way accept the prevalent opinion that choked disc is due to embarrassment of venous off-flow from the globe due to pressure on the nerve either by sheaths distended with inflammatory fluid exudate, solid mass, or edematous transudate, or to edema of the nerve itself, as from neuritis, and is accordingly purely or mainly of mechanical origin, while neuritis may be due to circulatory disturbance, to bacterial infection, to the action of toxins working at a distance from a purulent focus, to mechanical compression as in choked disc, or to a combination of some or all of these factors. For the practical purpose of the otologist choked disc signifies a morbid process which has resulted in a compression of brain tissue or at least in diminution of free space and so to a rise in intracranial pressure, while optic neuritis points to an inflammation of or in the immediate neighborhood of the meninges and an extension of this process either by continuity, or more probably by circulating bacteria or toxins, to the sheaths, and eventually to the substance, of the optic nerve.

The two conditions are dissimilar in symptomatology, clinical course, and to a certain extent in prognosis. Choked disc is characterized by protrusion, often amounting to several diopters, marked edema, generally marked dilatation or tortuosity of the veins, with narrowed arteries. Retinal hemorrhages are not infrequent. The nerve fibers become swollen and the nerve head appears made up of radiating striæ, often flecked with linear hemorrhages and resembling a chrysanthemum. There is no marked irregularity or obscuration of the disc margin. In extreme cases, of course, the intense congestion may induce slight inflammatory changes so that there may be a mild neuritis in addition. After relief of excess pressure in the cerebrospinal fluid a marked degree of choked disc may entirely disappear. Atrophy is an unusual sequela and is usually attributable to complicating neuritis or to intercurrent disease of the nerve or descending degeneration or inflammation from central disease. In neuritis, there is usually little or no swelling of the disc, so that protrusion is not noted. Hyperemia, on the other hand, is marked, and the outlines of the disc soon become indistinct or entirely invisible. This in marked contrast to papillary stasis, in which the disc is often enormously swollen and prominent, yet distinctly visible. The obscuration of the disc in neuritis may be so marked that the position of the nerve head is merely indicated by or rather to be inferred from the converging vessels at the center of the fundus. Marked changes in the caliber or course of the vessels are infrequent, almost exceptional. On the other hand, we note changes in the vessel walls in the form of perivascular striations, connective tissue proliferation starting at the disc and accompanying veins and arteries out along the fundus, as well as deposits of fibrin in the physiologic excavation of the disc, i. e., in the lamina cribrosa as well as at the margin. Of course, these changes are often obscured by the intense hyperemia and inflammation of the disc and by a fine opacity of the posterior layers of the vitreous and edema of the central part of the retina which almost invariably accompanies this condition. It is only in the later stages, particularly that of postneuritic atrophy, a frequent sequela, that these processes are made manifest by irregular outline of the disc, blocking up the lamina and physiologic excavation, and veiling of the markings of the cribrosa, perivascular connective tissue strands, and by pigment deposits on the disc or along its margins. Retinal hemorrhages are rare in uncomplicated optic neuritis. When present they may be due to choked disc, to vessel wall changes caused by mechanical obstruction of the circulation, or to vascular disease of a septic nature, as in pyemia or metastatic or septic retinitis.

In the construction of the table I have spoken, quite generally, of fundus changes, as opposed to the normal eyeground, in order to render the comparison of positive and negative findings more simple and graphic. In each case, however, the fundus conditions, whether neuritis or choked disc, and their distribution in one or other fundus or both, whether equally, or predominantly on one side, are indicated by the signs which are explained in the footnote. In the same table the cases are arranged in two main divisions, according as they resulted in death or recovery, so that it is possible to compare the frequency and severity of the fundus changes in mild and in fatal cases for every variety of intracranial complication. This, it seems to me, makes it much easier to get a general view of the subject than if we follow the example of Hansen and of Koerner and construct a number of tables dealing separately with the factors of distribution, severity, frequency and result. We are better able to judge of the prognostic significance of the presence or absence of fundus changes in endocranial complications if each form of disease is shown separately than if we make two categories, death and recovery, and compare the fundus findings in the two divisions for the entire number of cases. Instructive facts may be obscured by such figures which are actually out of place. For it is evident that figures which show a remarkably high percentage of fundus changes in fatal cases of, let us say, sinus thrombosis, will be balanced and lose their identity if combined with others showing indeterminate, negative or even opposite conditions in abscess or meningitis. The same considerations apply to a statistical study of the distribution of fundus changes, meaning the occurrence in one or other eye with greater frequency or severity, and the significance, if any, of bilateral changes.

A study of the individual and various forms of intracranial complications does not bear out, in my statistics, the conclusion of Koerner that the presence or absence of fundus changes is of no value in the prognosis of intracranial complications. The results as to distribution are also less ambiguous. Koerner found intraocular changes, without exception, in every instance, bilateral, and more often to a more marked degree on the sound than on the affected side.

EXTRADURAL ABSCESS.

In 6 cases of uncomplicated extradural abscess the fundus was invariably found normal. In 8 cases complicated with sinus thrombosis there were fundus changes in 3 (37.5 per cent.), all bilateral.

MENINGITIS.

In purulent leptomeningitis without other complications, changes of the nerve head were found in 4 out of 5 cases, all of which ended fatally. The changes were bilateral in all but one case, in which they were limited to the side corresponding to the mastoid involvement. In this case, choked disc was found, which in another was associated with optic neuritis of the other eye. Of 9 complicated cases neuritis was found in 6, slight changes in the vessels in 1, and a normal fundus in but 2. The intraocular disturbance was without exception bilateral.

SINUS THROMBOSIS.

In sinus thrombosis fundus changes were found much more frequently than might have been expected from the reports of previous statistics. Of 17 uncomplicated cases, no less than 12 showed decided fundus changes. All the fatal cases, 12 in number, showed fundus changes, while the 5 without fundus changes all recovered. In these cases, too, the intraocular disturbance was always bilateral, with the exception of the case reported by Gruening and included in my statistics, already mentioned, and one other, in which a thrombosis of the central retinal vein, probably of metastatic origin, was the only fundus change. In sinus thrombosis with extradural or perisinuous abscess, of 7 cases which recovered only 2 showed neuritis. Of the 2 fatal cases, one was normal, the other showing double neuritis. In all of this group, again, the fundus changes were bilateral, except in a case of general thrombosis of the sinuses complicated with meningitis on one side only, strange to say, and it was a question to which of the two intracranial complications the intraocular condition was to be attributed. The other cases of complication with meningitis are grouped under the latter heading above. Of the entire series of thrombosis cases, 28 in number, changes were found in 17 (61 per cent.). This observation is in marked contradiction with the figures of Hansen, who found them in only 37 per cent. of his thrombosis cases, and of Koerner, who had 5 uncomplicated cases, all but one with normal fundus. In view of these figures we must modify Koerner's conclusion that unilateral occlusion of the venous channels in lateral sinus and jugular does not seem sufficient in most cases to cause congestion in the ocular fundus.

BRAIN ABSCESS.

One case of uncomplicated temporosphenoidal abscess and 3 of uncomplicated cerebral abscess showed normal conditions in the fundus. In one case of uncomplicated brain abscess there was neuritis. Reviewing the combination of observations thus briefly reported, we may still say that changes in the fundus oculi may be absent in all and any forms of endocranial affection, but, and this is almost as unfortunate from the standpoint of differential diagnostics, they may also be present in any and all, with the possible exception of uncomplicated extradural abscess. An exclusive or more markedly unilateral ocular involvement is rare and generally without significance, but in sinus thrombosis it may be a valuable sign. The prognostic significance of fundus changes for the course of the intracranial complication is somewhat obscured by the fact that mild cases are often not examined ophthalmoscopically, while in the fatal cases the condition of the fundus is more apt to have been investigated at some time or another. It would appear that the absence of neuritis in thrombosis is of good omen, as it was not missed in a single one of my fatal cases, but the presence of neuritis does not make the outlook more grave, as there is about an equal percentage of recovery and of death after it has been found.

Dr. J. D. Richards, of New York, has been kind enough to give me the results of his own experience in this field. He has seen extradural abscess produce papillitis in three cases, in all of which it was on the affected side. What percentage this is he is unable to state, but thinks it must be exceedingly small. In sinus thrombosis it is probably not over 5 per cent. Richards has not seen the high percentage referred to by some writers as associated with thrombosis. A point of interest in the sinus cases to which Jansen and he have called attention is that those cases which showed neuroretinitis had the jugular thrombosed. In these cases the inferior petrosal is also blocked and the damming back of venous blood upon the cavernous sinus and thus upon the ophthalmic vein and intraocular vessels is probably responsible. He believes that when we have a sufficient number of observations to enable us to draw definite conclusions, this agency of jugular bulb thrombosis in the causation of fundus changes will be corroborated. At least three of the cases collected by me seem to support this view. In one (43) there was a thrombus in the lower part of the sinus only, probably implicating the bulb, and double optic neuritis. In another (67), ending fatally, there was neuritis on the thrombosed side and later positive evidence of cavernous sinus involvement in the form of edema of the eyelids, beginning on the affected side and afterwards involving both eyes. In a third case (56), the sinus was apparently normal at operation. A peaked temperature curve, pressure pain along the jugular, and slight neuritis, more marked on the affected side, led to evacuation of the sinus and excision of the jugular vein, followed by recovery. In this case it would seem that there was a primary thrombosis of the vein, which at the time the mastoid was opened had not yet involved the upper part of the sinus. The conditions were almost paralleled in Gruening's case, which I have included in my statistics, as the fundus changes were found only on the affected side, and the sinus appeared healthy not only at the primary operation, but at the time of jugular extirpation. On incision, however, it was found to contain an infected clot, so that here, too, it seems quite probable that there was a primary bulb thrombosis with very little tendency to advance upwards, but with rapid infectious softening.

Comparing the results of his own investigation with those of Hansen, Sessous and Koerner, the writer has but little to add, except in corroboration. The absence of fundus changes in a given case has no significance. Its presence, on the other hand, is of the greatest importance. This bearing is not strictly diagnostic, as we can not draw any conclusion from the presence of fundus changes as to the nature of the intracranial affection, but it is an absolute indication of the presence of some complication, and as such makes it advisable, if not imperative, to explore the cranial cavity and, if possible, remove the source of suppuration and of local as well as systemic infection. To put the case in a nut-shell, fundus changes may be absent in intracranial complications, but intracranial complications are never absent with fundus changes. The otologist should make his own ophthalmoscopic examinations. This does not imply that an ophthalmologist is not to be consulted in a doubtful case and the observations compared. Early and repeated examinations in each and every case can only be assured by the ability of the aural surgeon to use the ophthalmoscope with skill and experience. Ophthalmoscopic examination is usually neglected unless the previous history or the development of other symptoms of intracranial involvement directly suggest it. Early and repeated examinations would add greatly to the value of our statistics and to our knowledge of the pathogenesis and significance of fundus changes, and routine examination on admission of all operative cases would not only supply much valuable material, but avoid the loss of important observations due to the frequent impracticability of carrying out an ophthalmoscopic examination on the sudden development of ominous symptoms, the preparation for further urgent operation, or, indeed, sudden death.

In addition to the observations on fundus changes, I wish to report quite briefly on one or two other ocular symptoms. In a comparatively large number of cases of generally uncomplicated otitis or mastoiditis, edema of the eyelids limited to the affected side was found as an expression indicative of a marked involvement of the zygoma cells (1, 9, 15, 29), complicated in 2 cases (9, 29) by subperiosteal abscess. The same symptom was found in 3 cases of secondary inflammatory infection of the soft parts, after removal of all diseased bone (25, 26, 29) in ordinary furunculosis of the external auditory canal (14, 63) and in stitch-hole abscess developing a week or two after operation (10, 23). In two cases, finally, this edema of the lids was due to conditions which had no connection or only an indirect connection with the otitis, as erysipelas (71) and frontal sinusitis (11). Lid edema as a symptom of intracranial complication was observed in 3 cases. Once in spontaneous recovery from a probably incomplete sinus thrombosis (21), again bilaterally in sigmoid sinus thrombosis with thrombosis or stasis in the cavernous sinus (67). Ophthalmoplegia occurred in 2 cases of brain abscess (2, 19), and, again, as an expression of mechanical fixation of the globe by inflammatory products in the orbit, in thrombosis of the cavernous sinus (38). Convergent strabismus was noted in meningitis (72), meningitis with sinus thrombosis (64), and meningitis with epidural abscess (32, 57, 59). Abducens paralysis occurred in one case of uncomplicated (?) middle ear suppuration, and once, probably as a result of traumatism, after exploratory incision of the brain (13), once in combination with paralysis of the trochlearis in a case of brain abscess with meningitis (30), and in one case, finally, of meningism following sinus thrombosis (55). Conjugate deviation of the eyes, as well as nystagmus, was found in several cases of brain abscess and meningitis, and in one of lateral sinus thrombosis, but, as the case histories and autopsy records, respectively, of these cases did not completely explain the symptoms or establish the diagnosis on a pathologic-anatomic basis, they will not be condsidered further.

BIBLIOGRAPHY.

1. Archives of Otology, Vol. viii, 1879, p. 147.
2. Ibid., Vol. xiv. 1885, p. 1.
3. Diseases of Ear, 1902.
4. Encyclopedie d. Ohrenh. Art., Auge, Ostmann, 1900.
5. Diseases of Ear, 1901.
6. Woodward, Arch. f. Otol., January, 1906.
7. Lehrb. d. Ohrenh., 1906.
8. Otogene Erkrank. d. Hirn., 1903.
9. Ohrenh., 1904.
10. Diseases of the Ear, etc., 1906.
11. Diseases of the Ear, 1905.
12. Otit. Erkrank. d. Hirns, etc., 1902.
13. De Schweinitz and Randall. System; Complications of Tympanic Inflammation, 1899.
14. Diseases of the Ear, 1903.
15. In addition to this material I have included all cases reported in the last (1906) volume of Transactions of the American Otological Society, of Intracranial Otitic Com-

plications in which an ophthalmoscopic examination was made. They are one case of lateral sinus thrombosis (Gruening), and two cases of cerebellar abscess (Dench, A. Knapp). Gruening's case was particularly interesting as there was bilateral mastoiditis with exposure of both sinuses at operation. Characteristic temperature changes arousing suspicion of thrombosis, an examination of the fundus was made and hyperemia of the discs found on both sides. On only one side, however, did this progress to optic neuritis, and on the strength of this finding, and in spite of the absence of evident local changes in the sinus wall of this side, and the presence of some discoloration on the other, the apparently healthy sinus was opened and found to contain an infected clot. Here the diagnosis was made by the ophthalmoscope alone, not only unsupported, but actually contradicted by the local signs.

16 July, 1907.

IV.

A CASE OF THROMBOSIS OF THE POSTERIOR CEREBRAL VEINS AND ARTERY, ENCEPHALITIS, PURULENT LEPTOMENINGITIS, LATERAL SINUS PHLEBITIS AND PARIETAL THROMBOSIS FOLLOWING MASTOIDITIS.

By W. Sohier Bryant, A. M., M. D.,

New York.

A well-nourished woman 50 years of age was seen by the writer in consultation at the Manhattan State Hospital. She had a history of having had grippe more than four weeks previously, since which her mind had become unbalanced. The physical condition of the patient had been of negative interest except for diminished thoracic expansion, up to the day her temperature began to rise without any apparent cause. Three days previous to the rise of temperature her physical examination was negative. Her ears showed nothing abnormal.

On the fourth day after the commencement of the rise of temperature, rales and scattered patches of exaggerated breathing were noted at both bases behind. The right ear began to discharge thin pus. On the sixth day of the illness, the patient appeared very sick. Discharge from right ear continued. Some tenderness behind ear and indefinite earache. Lungs—dullness, rales, and exaggerated respiratory sounds over lower half of both lungs behind. Heart sounds very weak. On the seventh day the patient appeared better. Discharge from the right ear had ceased. No mastoid tenderness and little aural discomfort. Lungs slightly improved. Hemoglobin, 70 per cent. Leucocytes, 22,000. Ear irrigation continued.

On the eighth day the author did a complete mastoid operation for acute mastoiditis. Free myringotomy. The mastoid was opened in the usual way by first removing the tip, which appeared perfectly normal. The mastoid was very cellular, especially inferiorly and posteriorly, where the cells were filled with granulations, and there was free bleeding. Only two minims of pus were found which was located over the region of the lateral sinus far back. The bone covering the sinus appeared healthy. The sinus was not exposed. The dura mater of the middle fossa was exposed through the tegmen antri posteriorly. It was very slightly congested and had slightly increased tension. The wound was washed and closed over a small drain. Iodoform gauze was put into the canal and a wet saline dressing outside. Time consumed, 35 minutes. Took anesthetic well. Returned to bed in fair condition. Examination of the pus from the cells showed large diplococci. Bacterial examination of the blood was negative.

The ninth day of the illness or first day after operation: Radial pulse very weak. Lungs apparently unaffected by the anesthetic. Leucocyte count, 20,600. Second day: Examination. Pupils small, regular and active. No stiffness of neck, Knee-jerk slight on right, absent on left. Achilles reflex present on left, absent on right. Some tendency to Babinski sign in left great toe. Spinal puncture, 5 cc. slow flowing, clear fluid withdrawn; no blood. Microscopic examination showed encapsulated diploccoci similar to those found in the mastoid pus, staphylococci and a few bacilli and blood cells. No polynuclear leucocytes. Examination of the blood culture negative. Heart regular and rapid. Lungs unchanged. Patient dull, restless, no pain. No motor symptoms observable. Patient gradually became weaker, lungs more edematous. Radial pulse entirely lost. Temperature ranging from 105° to 106° followed by death on the eleventh day of illness and the third day after operation.

AUTOPSY BY DR. GLANVILLE Y. RUSK.

"Remains of a moderately well-nourished female. Pupils, circular, equal, moderately dilated. The amount of cerebrospinal fluid moderately increased. Over the right convexity, the subdural space shows a puriform cerebro-spinal fluid and the convexity, especially along the Sylvian fissure, is covered with a somewhat granular fibrino-purulent exudate; the exudate is for the most part in the meshes of the pia-arachnoid or strips off with it, except near the anterior end of the temporal fossa, where for an area about 3x5 cm. the exudate is closely adherent to the dura. The veins over the right hemisphere are more prominent than over the left, the large veins over the temporal lobe and along the Sylvian fissure being the most marked. On opening the lateral sinus, it is found to contain an adherent thrombus, starting anteriorly at the junction of the superior petrosal sinus with the lateral sinus and extending backward about 5 cm. along the superior and outer wall of the lateral sinus. A thrombotic plug is also present at the mouth of the jugular foramen. The point of most adherent pus formation to the dura lies about an area approximately 5 mm. in diameter, where the overlying bone has been removed during operation, and which is situated about 2 cm. above and slightly behind the auditory meatus. Smears from the intracranial pus show streptococci in short chains. Sections from the cortex show typical leptomeningitis with occasional streptococci, the inflammatory process not invading the brain substance. Unusually good examples of phagocytosis are present. The large nerve cells show examples of acute alterations of moderate grade."

Mastoid wound in good condition, clean and healing by first intention. The parietal thrombus of the lateral sinus covers and enters the mouths of the posterior cerebral veins. Thrombosis of right posterior cerebral veins and artery. Thrombus of jugular foramen appears to be more recent than the parietal thrombus of the lateral sinus. The meninges of the cerebellum and left hemisphere, normal. Ventricles, normal. Lungs filled with fluid. Death due to mastoiditis and pulmonary edema.

Capitulation.—The author was unable to find any references in literature to a similar extension of infection from the mastoid cells. The case seems to be unique in this respect that the infection from the mastoid crossed the lateral sinus without encroaching upon its lumen to any extent, entered the posterior cerebral veins and caused a fatal leptomeningitis which was out of all proportion to the comparatively slight mastoid involvement which was obscured by the concomitant pulmonary signs.

An interesting point shown at autopsy was the presence of two separate and distinct thrombi of different age, on the same side; one, of the lateral sinus; the other, of the jugular foramen.

The differential diagnosis between the complicated mastoid affection and lung affection was aided by the lack of parallelism of the respiration, pulse, and temperature curves and by the improvement in the condition of the lungs independent of the amelioration of the septic condition. In this case there was no macroscopic connection between the mastoid infection and the meningitis.

The temperature curve was not characteristic of sinus thrombosis since the rises were maintained for a time, blunting the characteristic saw-teeth. After the mastoid operation the question of jugular ligature was not raised because the temperature curve did not indicate thrombosis. The character of the old thrombus found at autopsy was not the kind likely to give rise to a characteristic temperature curve. The question of other intracranial explorations was considered and was decided against because of extreme weakness of the circulation. The autopsy showed the certain futility of any such procedure owing to the extensive leptomeningitis.

The history has been compiled from the hospital record kindly furnished by Dr. William Mabon, from the pathologic notes contributed by Dr. Glanville Y. Rusk, and from the author's own clinical and autopsy notes.

V.

A PLEA FOR CONSERVATISM IN THE TREATMENT OF CERTAIN AFFECTIONS OF THE NOSE AND EAR.

By Harry L. Myers, M. D.,

Norfolk, Va.

Some one has aptly said "it is better to be conservative in radicalism than radical in conservatism." The difficulty in this age of advanced surgery seems to be in finding the safe channel between the Scylla of conservatism and the Charybdis of radicalism. I fear that I shall not be able to point out the way in these troubled waters, but I hope to create a discussion at this meeting by which I may learn the better to avoid the dangerous rocks on either side.

With greater experience, I believe that there has arisen a spirit of conservatism among us which will continue to grow. The literature of the day confirms this and, recently, my courage to present this subject has been not a little strenghtened by articles from some of our best men, expressing their belief in conservative methods of treatment in diseases of the ear and nose. It seems to me that it is in the surgical treatment of nasal diseases especially that more conservatism is needed. The antrum of Highmore, owing to its size, peculiar shape and dependent position, is not only particularly liable to infection but most frequently suffers from involvement of the sinuses situated above it, collecting their secretions which pour down, thus sometimes acting as a reservoir and assuming the appearance of guilt when it is really innocent.

The operation upon this sinus through the inferior meatus marks, in my opinion, one of the greatest advances in the surgery of the nose and is really, compared to the external operation, a victory for conservatism in that it is now used almost exclusively in work on this sinus, except in those cases in which there is sufficient necrosis to demand an external opening. I, however, think that even this operation, on account of its ease of performance and the lack of the necessity of general anesthesia in many cases, is apt to tempt us to destroy a large piece of the inferior turbinate and a goodly portion of the inner wall of the sinus when frequently we could accomplish a cure by puncture through the inner wall of the sinus under the turbinate, and irrigation of the cavity every other day with a bland alkaline solution, over a period of three weeks or two months. Many cases, which have extended over months and whose discharges were decidedly offensive, have yielded to these mild measures at my hands. I do not mean to say that the cases I have been fortunate with were all cured by irrigation alone. When there is obstruction around the ostium of the sinus, whether it be from enlarged turbinate, deviated septum, polypi, or disease in the contiguous ethmoidal cells, I have removed these obstructions. In fact, I may say that in nearly all except acute cases, and in some of these, I think it will be necessary sooner or later to remove the anterior end of the middle turbinate. This is, however, a conservative measure and does not seem to do other than good. I am convinced that were more work done on the middle turbinate in these cases, and less on the inferior, results would in the end be better. It has been pretty well proven that the inferior turbinate has a well defined place in the moisture producing function of the nose, and I never could persuade myself that any more of it than absolutely necessary should be removed.

Before resorting to this operation we should be as certain as possible that the antrum is not acting simply as a reservoir, as under such circumstances we should fail to accomplish our end, and at the same time would produce an abnormality to no purpose. In most cases of chronic discharge from the antrum there will be found disease in other sinuses above it, and these sinuses should be treated and their secretion stopped, if possible, before more than irrigation is done to the antrum. Those cases of antral discharge where the trouble can be traced to diseased teeth, generally yield to drainage and irrigation through a generous opening in the tooth cavity, after the offending member has been withdrawn. Greater patience, however, is required in these cases, as time must be given for nature to throw off the necrotic tissue which may have formed in the floor of the antrum, around the diseased root. I may say here that I have experienced no trouble with food getting in through the opening in the alveolus, and I neither use an obturator nor a tube. I have experienced little trouble in the cavity closing, as I direct the patient to use a Moffett's dental syringe which has a conical canula with a rather sharp point, thus being easy of introduction and serving to push back the parts effectively and without pain. Where there is much necrosis I believe the older operation from the outside is necessary.

We of the South have fewer cases of disease of the frontal sinuses with which to deal; most cases which have come under my observation have been rendered comfortable and in many cases cured of pain and discharge by conservative measures within the nose. I find that the removal of the anterior end of the middle turbinate and other obstructions to the infundibular opening, together with irrigation, has been of such service that few of my patients were willing to have resort to the external operation. I have never tried the method of boring into this sinus from the nose, and I never shall, as I consider it impossible for any one, however skillful, to know where the end of his drill is going, and as between this method of treatment and that of an external opening, the external method is the more conservative. I think, however, one may accomplish much by the cautious use of the curettes in enlarging the canal in an anterior direction, as recommended by Hurd; where there is bulging of the eye or tissues, or where pain is not relieved by conservative internal methods, the external operation often becomes a necessity.

I recognize that I am treading on dangerous grounds when I attack some of the methods used to-day in dealing with septal deviations, but I am afraid we are sometimes too bold in the use of the "window" resection operation. First, let me say that I feel that within proper limits it is the greatest advance in nasal surgery, and it will eventually be the principal method used for corrections of septal deformity. To my mind the danger of it lies not in its use, but its abuse. The more skillful we become in its technic, the more anxious we are to remove every bump and projection; in other words, we strive for a septum that looks perfect. In our enthusiasm for such a result I believe we not only at times remove more tissue than is necessary, but sometimes more than is safe. It seems to me that what we should strive for is to remove as little as possible in order to restore good nasal breathing regardless of appearances. We all know of very crooked septa where nasal breathing is perfect, and a projection which does not obstruct should be left alone. I am also of the opinion that we cannot yet afford to give up the nasal saw, for with it we can many times accomplish as good a result and we certainly feel more certain of the contour of the external nose.

The treatment of hypertrophic rhinitis has made advances in late years, due perhaps no little to an improved armamentarium and the estabished view that it rarely does harm to remove portions of the turbinate bodies with scissors and such instruments.

The careless use of the galvano-cautery has been so criticised that we are in a fair way to lose a very good means of relief in mild cases of this trouble; a means that is being used to-day by men of undoubted experience, and whose results are second to none. In those cases where there is no great thickening of the soft tissues but where the patient complains of great stuffiness at times, and in cases of intumescent rhinitis, where the patient either will not or cannot live an out of door life, I have found linear burns along the inferior turbinate with either the galvanic or chemical cautery to give me most excellent results, and I do not believe our enthusiasm for the newer cutting methods should lead us to abandon entirely the more conservative method in selected cases. Where there is great hypertrophy or deformity the snare or scissors have given me better results, but even then I think as little as possible of the bony turbinate should be sacrificed, except where the middle turbinate is concerned, and here I believe the cautery should never be used.

In chronic suppurative diseases of the middle ear, the time to relinquish conservative measures for radical treatment must necessarily depend upon several conditions. Environment, for instance, must be considered, for when a patient lives within easy reach of a competent aurist and has the means to obtain skilled treatment, he, in the absence of urgent symptoms, may frequently be made comfortable and rendered reasonably safe by the occasional use of intraaural methods of treatment, and I am of the opinion that we are scarcely justified in operating upon such a case radically when about the only symptom of annoyance is more or less discharge from the ear. On the other hand, the patient who is suffering from a chronic aural discharge with occasional acute exacerbation, who must live far removed from skillful treatment, had best submit to the radical operation.

Another condition which would indicate the necessity for radicalism is an unconquerable offensive discharge which in some cases renders the life of the patient most miserable. When the clearing out of the tympanic cavity from the meatus either with the curette, chemicals, syringe or the operation of ossiculectomy (which, in my experience, while not always causing complete cure has never failed to improve) does not relieve pain and furnish good drainage and fails to greatly relieve all other symptoms, the radical operation should be considered.

When the probe demonstrates necrotic bone in the attic and when cholesteatomatous material is present in the discharges, we must sooner or later resort to radical measures.

In a report recorded some time ago by Dr. Harris before this society, he showed that during ten years in one of New York's institutions for treatment of ear diseases, out of 32,000 odd cases of ear diseases there were only 60 cases of intracranial involvement. In the same report he shows from statistics of many authorities that intracranial involvement was very rare.

Koerner made the statement that the radical operation is indicated as soon as a diagnosis of chronic purulent otitis media is positively made, but he also states that he has never seen or heard of a case of semi-mucopurulent discharge from the antrum, without obstruction to the outflow, that resulted in intracranial disease. In my own experience, extending over a period of thirteen years, I have seen meningitis once in a chronic case, serous meningitis once in an acute case, sinus thrombosis five times in acute cases, and twice in chronic cases. Since intracranial complications seem to be rare I have felt that we should employ every possible conservative means of relieving these cases before resorting to radical measures, which often not only fail to cure, but too often renders the patient's life more miserable.

VI.

PRIMARY NASAL DIPHTHERIA.

By Thomas Hubbard, M. D.,

Toledo, Ohio.

Fibrinous rhinitis is a suggestive descriptive name for the disease under consideration, and like membranous rhinitis serves to link the old with the new pathology of diphtheria; but as membranous croup has given way to the more scientific expression, diphtheritic laryngitis, so will this type of rhinitis be called primary nasal diphtheria.

There is no doubt a form of membranous rhinitis, with exudate or exfoliation, resembling the true coagulation necrosis of diphtheria, caused by various bacteria, chiefly cocci, but these conditions are so rare that they may be eliminated from consideration.

Primary nasal diphtheria is a manifestation of the disease not generally understood by practitioners, and there is lack of clear forcible descriptions in some of the text books devoting many pages to diphtheria.

In passing I would make the criticism that we are less careful in the matter of diagnosis than in the early antitoxin period —that is, we do not utilize all of the aids to early accurate diagnosis. As the specific antitoxin has grown in favor we have become content to depend on the therapeutic test. It is all very well to say: "In case of doubt give antitoxin," but the trouble is that in laxity of diagnostic zeal we forget to doubt. I have seen primary nasal diphtheria overlooked so often by general practitioners that I feel justified in presenting clinical experiences and opinions of writers on the subject.

As compared with faucial diphtheria of the ordinary type, primary nasal diphtheria differs in two important features. It is nearly always a subacute or chronic disease process, developing insidiously, and the element of toxicity is comparatively slight. These two clinical characteristics we should keep clearly in mind.

Diagnosis is often difficult, sometimes impossible, without the aid of the laboratory expert. False membrane may form in any part of the nasal fossae or nasopharynx, or we may have diphtheritic rhinitis with very little or no coagulation necrosis, only a hyperemia with a catarrhal flux. These are extenuating factors in tardy diagnosis, but even in obscure manifestations there are always suggestive clinical symptoms which should lead the careful observer to have made a laboratory diagnosis or make a timely therapeutic test by administering antitoxin.

Nasal diphtheria secondary to faucial diphtheria presents a different clinical picture. It is an extension of the acute disease process, with marked increase of toxemia, and rarely escapes diagnosis. It is mentioned only to exclude it from discussion.

There are two types of primary nasal diphtheria. In the one there is a formation of false membrane, sometimes to the extent of completely filling one or both nasal fossae with fibrinous casts, and a watery, acrid, mucopurulent discharge; and the other is merely a diphtheritic inflammation of the mucosa with a copious watery discharge of acid muco-pus. Altered blood pigment may give a reddish or brownish tinge to the secretion, and the nostrils and upper lip are usually excoriated. As a rule we find the fibrinous type in older children, and not rarely in young adults; and the catarrhal type is charateristic of primary nasal diphtheria in infants. In both, as previously stated, the disease runs a subacute or even chronic course, with very slight toxemia considering the area of tissue involved, and low mortality except in very young infants in whom mild sepsis and nasal obstruction interfere with nutrition. The disease as thus manifested in the nares is in a degree self limited but may run a course of several weeks, and there is a tendency to reinfection and relapse.

Extension from nares to fauces is rare and this is one of the puzzling clinical features. It is astonishing to see such an extensive area of infection with natural drainage into the throat and no manifestations of faucial diphtheria to be seen. Holt states that extension downward does take place, but ususally in cases having the more acute type of primary nasal diphtheria. It rarely occurs in the chronic fibrinous type.

Another remarkable clinical feature of the chronic fibrinous type is that one naris may show evidences of extensive false membrane deposit and the other be free from visible evidences of infection. The discharge may be from one nostril only, even in the advanced stage.

Culture tests show frequently pure Klebs-Loeffler, and while it is not proven that these are especially virulent, indeed I suspect from clinical experience that they are less than ordinarily virulent, yet the facilities for transmission are so much greater than in faucial diphtheria, the victim rarely being ill enough to be isolated or confined to one room, constantly using the family towel and drinking cups, and spreading the infection by contaminated fingers, that the net result is widely disseminated infection, and the most virulent type of faucial diphtheria may result in those infected.

This leads up to a more concise grouping of symptoms which should arouse suspicion. Simple acute coryza is the mask behind which primary nasal diphtheria invades the ranks of infancy and childhood. A watery, acrid, purulent discharge from one or both nostrils should always excite suspicion of primary nasal diphtheria, and if examination does not show false membrane the laboratory expert should be sent for and a culture test made. A foreign body produces similar symptoms but usually diagnosis is easy.

The systemic symptoms are, as stated, very mild and attract little attention. A degree or two of fever for a few days, malaise and anorexia, restlessness at night, due partly to nasal obstruction, are the principal symptoms. Toxemia is not very marked but there may be sequellae, paresis of groups of muscles as in ordinary faucial diphtheria. I have found albuminuria in several cases in which there were very slight systemic symptoms. There is often slight enlargement of cervical glands, not due necessarily to the diphtheric process but in part to mixed infection and local sepsis.

Primary nasal diphtheria in infants merits most careful study and it is often so obscure that it escapes detection even in institutions devoted to the care of infants.

I will quote from the address of Dr. John L. Morse (assistant professor of pediatrics, Harvard, assistant physician Children and Infants' Hospital):

"Membranous rhinitis is much more common in infancy than is usually supposed. It is generally of a very mild type and is usually not suspected until other children come down with pharyngeal or laryngeal diphtheria, and often not even then.

It is, perhaps, the most common source of the epidemics of diphtheria which develop in hospitals and institutions for infants and children. My experience at the Infants' Hospital has taught me to suspect every nasal discharge in an infant, especially if it is irritating.

"In spite of the greatest care in the admission of cases we found it imposible to avoid epidemics of diphtheria in the hospital from this cause until routine immunization was adopted. The babies now receive 500 units of antitoxin on admission and every three weeks afterward. Since this practice was begun, six years ago, but one case of pharyngeal diphtheria has developed in the patients in the hospital, although many unimmunized nurses and nursery maids have come down with it, and many cases have been discovered.

"The symptoms of nasal diphtheria in infancy are, as a rule, very mild. There is usually a little snuffles with a slight nasal discharge and a little nasal obstruction. There is no glandular enlargement and the constitutional symptoms are wanting or very mild. The nasal discharge, however, is often rather characteristic, being thin, watery and acrid, but, contrary to the usual statements, rarely bloody. It frequently irritates the upper lip, causing crusts about the nares and on the lip. A thin, watery, nasal discharge, which irritates the lip, should, in an infant, always be examined bacteriologically. The amount of the discharge varies markedly in different cases. There is almost never any membrane visible unless the speculum is used, and even then it may not be seen. The diphtheritic process practically never spreads backwards and downwards to the pharynx and larynx. The diagnosis must depend, therefore, in most cases, on the bacteriologic examination of the nasal discharge."

One of my earliest cases was in a family of seven children. The five-year-old boy had diphtheritic laryngitis, dyspnea so extreme that he was unconscious. I intubated and respiration ceased altogether. Tracheotomy and artifical respiration for 15 minutes restored consciousness. I then looked over the family and found the culprit. A lad of twelve had an excoriated upper lip from an irritating watery, purulent discharge from both nares. A small deposit of false membrane was seen on the septum. He had been in this condition for more than ten days, and without the slightest throat symptom or effect on his general health, and had been daily in school.

Administered antitoxin to all of the family. Within 24 hours three children had faucial diphtheria and two more had diphtheria of larynx, but large doses of antitoxin saved them from intubation. All recovered. The discharge from the primary nasal diphtheria case showed Klebs-Loeffler bacilli.

In one experience I had to combat the opinion of the family physician, a man of wide experience, as he was opposed to the administration of antitoxin for what he termed purulent catarrh. The lad was apparently in good health although there had been a watery discharge from both nostrils for more than a week.

He came to me on account of nasal obstruction, and I discovered large plaques of firm white membrane on both sides of the septum. In this case I administered 6000 units of antitoxin and soon had confirmation of diagnosis by report of bacteriologist. Within 48 hours a younger brother and sister had severe faucial diphtheria and soon after the mother and an aunt had the disease. A baby of 12 months was given an immunizing dose and had only catarrhal symptoms, possibly mild nasal diphtheria.

Five or six cases of primary nasal diphtheria have been brought to my office and nearly always the diagnosis "foreign body in the nose" has been suggested by the family physician. In fact, two had undergone operation for removal under general anesthesia. This error is referred to by Northrup (Nothnagel's System of Medicine) as being frequently observed in dispensary service.

Frequently primary nasal diphtheria runs its course undiagnosed, and a tell-tale sequela, in the nature of paresis of certain muscles, reveals the true nature of what was considered coryza. A young man of 17 returned from boarding school in apparently good health to spend a vacation. It was noticed that the voice had a peculiar nasal character, and he swallowed fluids with difficulty. Examination showed one-sided paresis of the velum palati, and an excoriated septal mucosa, posterior third, with small plaques of false membrane. Bacteriologic examination showed Klebs-Loeffler bacilli. This was about three weeks after he had apparently recovered from what was termed coryza by the dormitory physician, although at the same time one boy was sent home with tonsillitis.

This case was unusual in that he did not have the characteristic acrid nasal discharge, the disease being limited to an area involving the posterior third of septum on one side only, and it was of decidedly chronic character. No antitoxin was administered and the throat paresis gradually subsided.

In another similar case, a child of three, the diagnosis was not very positive, no bacteriologic examination having been made, but the therapeutic test was decidedly confirmatory. Paresis of velum developed in two weeks. In these two cases, and in others, I have been impressed with the lack of virulence of the infection, as evidenced by the chronic course of the disease, and by the fact that many other children in close contact with the one sick did not contract the disease.

Concerning the degree of virulency of the bacilli in these cases of the chronic type, Dr. Thomas B. Cooley of Detroit makes the observation, based on an extensive experience in tthe contagious disease wards of the Boston City Hospital, that the presence of the copious sero-mucus discharge probably has an inhibitory influence on the growth of bacilli, and thus effects the virulency, but one cannot draw any positive conclusions. A given case of very mild chronic type may produce an acute fulminating type of faucial diphtheria in another individual. Dr. Cooley confirms the opinions of other writers, that primary nasal diphtheria, from the fact that it is so often overlooked, is the most potent factor in the causation of epidemics. In his experience it is very difficult to find false membrane in infants and he would depend more upon the character of the nasal discharge. Contrary to the opinion expressed by Morse, Dr. Cooley has often found altered blood pigment in the discharge from the nares, and especially in infants

The treatment is essentially the same as that of faucial diphtheria, except that all writers are agreed that in typical primary nasal diphtheria relatively smaller doses of antitoxin seem efficacious. It is astonishing how quickly all symptoms melt away after a dose of 3000 units. But yet, considering the fact that diagnosis is made late, as a rule, I am in favor of a rather large primary dose, 6000 units in first 24 hours.

The temptation is to attack the local condition with antiseptic irrigation or douching, but all are agreed that it is futile and not without danger to accessory sinuses or the ears. A mild detergent solution dropped into a nares with a medicine dropper is sufficient.

It should always be borne in mind that these cases of primary nasal diphtheria harbor bacilli longer than the ordinary faucial type, and the period of isolation should be prolonged.

VII.

A PLEA FOR LOCAL ANESTHESIA IN OPERATIONS ON THE MAXILLARY SINUS.*

By Gordon King, M. D.,

New Orleans.

In resorting to surgical procedures for the relief of conditions which, in themselves, do not entail risk to the patient's life or interfere so seriously with comfort as to render life burdensome, it becomes the prime duty of the surgeon to minimize in every way possible the risk to life incurred by the operation itself consistent with analgesia, avoidance of shock and thoroughness of execution.

How doubly unfortunate does it seem when a fatality occurs from the use of a general anesthetic in the performance of some simple operation for a minor ailment, for which, if operation is essential for relief, a local anesthetic skillfully and judiciously employed would have been sufficient to prevent pain, and at the same time would have been comparatively free from danger to life. Few of us but can recall to mind in the sphere of his observation or experience some catastrophe of this kind for which vain regrets offer but poor consolation.

Recognizing as we must, the ever present danger in any general anesthetic and the impossibility always to foresee and to prevent a possible accident, it is but just that every patient whom we are to subject to this danger, or those responsible for his welfare, should be made aware of it, however slight the risk may appear to be. Among the more intelligent we take it for granted that the risk is understood and accepted without discussion, but often the surgeon is anxiously interrogated as to any possible danger to be incurred, and in such instances it has always been my custom to express my opinion frankly, although regretting the undue anxiety this may cause. For these reasons and others of practical bearing, I have within the past few years endeavored to restrict as much as was practicable my use of general anesthetics and resorted more to local.

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*Read before the twenty-ninth annual meeting of the Americal Laryngological Association, Washington, D. C., May, 1907.

I have proved to my own satisfaction that in many cases in which formerly either through precedent or habit I relied upon general anesthesia, I now can accomplish equally good results under local anesthesia.

Especially is this true of operations about the mouth and upper air passages, where in addition to the danger from the anesthetic we have to contend with the annoyance and delay incident to nausea and vomiting, the inhalation of blood and secretions into the larynx, and the frequent interference with the administration of the anesthetic. These annoyances so often hindered me in antrum operations, where more than half the time is occupied with the administration of the anesthetic and delay caused by retching and the accumulation of blood and mucus in the throat, that I decided some time ago to try the efficacy of local anesthesia. The result has been even more satisfactory than I anticipated, and for two years past I have used a local anesthetic exclusively in these operations.

The antral region is peculiarly well adapted to local anesthesia, and even the sting of the hypodermic needle can be obviated by a preliminary swabbing of the mucous membrane in the canine fossa with a ten per cent solution of cocain.

Being still a loyal advocate of the Caldwell-Luc operation for antral empyema rebellious to the less radical treatment, I shall describe briefly the manner in which this operation can be performed practically without pain and in less than half the time required when a general anesthetic is employed. Including the first few minutes expended in benumbing the mucous membrane of the canine fossa by topical application, the entire procedure has on an average taken not more than seventeen to twenty minutes.

I begin by swabbing the mucous membrane briskly with a ten per cent cocain solution, then when the part is insensible to the prick of the needle a solution of cocain (2 per cent) and adrenalin chlorid (1-2000) is injected along the line of incision, at first superficially into the submucous tissue and then more deeply into or under the periosteum. This done, the incision is made down to the bone and the periosteum elevated until the exact situation of the infraorbital foramen can be made out, when to insure more perfect and widespread anesthesia a few drops of the solution are injected into or around the trunk of the infraorbital nerve at its point of exit.

VIII.

CONTRIBUTION TO THE STUDY OF TUBERCULOSIS OF THE NASAL MUCOSA—AN ETIOLOGIC AND CLINICAL STUDY.*

By Henri Caboche.

Translated by Albert Miller, A. B., M. D.

Clinical opportunities having given me occasion to see several cases of lupus and so-called tuberculous tumors of the nasal mucosa, I was soon persuaded that the classic scheme of tuberculosis of the pituitary membrane did not probably correspond with the facts, and I conceived the idea of reviewing the works upon which it is based.

In looking over the different works one is struck by the varying classifications of these writers.

Cartaz in his epoch-making memoir[1] and Boutard in his thesis[2] described two forms of nasal tuberculosis: one, usually primary, resembling a tumor; the other, usually secondary, taking the form of a tuberculous ulcer, clinically resembling other ulcerations of the mucosa. Further, ulcer and tumor may coincide.

This is the description which we again find in our classics (Lermoyez, Garel, Moure).

Plicque[3] describes tuberculous granulation, ulceration and tumor. The granulation, which is rare, is gray granulation which later gives rise to ulceration.

Olympitis[4] in his thesis inspired by Tissier, distinguishes no less than eight forms of pituitary tuberculosis which he divides into: Acute miliary, polypoid, infiltrated, lupus, tubercular ozena, submucous abscess, ulcerous, caseous. The last two as well as the acute miliary form are secondary to pulmonary localization, the others are primary.

Koschier[5] in a very complete and interesting memoir gives twenty-one personal observations of nasal tuberculosis. He distinguishes an ulcerative form, a nodular or neoplastic form, and an osseous and cartilaginous form, histologically and clinically analogous to tuberculosis of other bones.

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*From the Annales des maladies de l'oreille, etc., Oct., 1907.

Chiari (Affections of the Nose, translation by C. Breyre, 1905) distinguishes a chronic catarrhal form resembling ozena, an ulcerous form, and a neoplastic form.

All these writers distinguish tuberculosis, properly so-called, from lupus of the pituitary membrane, but they do not make clear how to determine clinically what is lupus and what is tuberculosis.

Cartaz, Boutard, Pistre[6] include neoplastic formations in tuberculosis; Raulin,[6] on the contrary, places them in the lupus class.

Similarly, in Germany, Schoeffer considers his six cases of tumors as the clinical manifestation of nasal tuberculosis, on the grounds that they were not accompanied by any lupic cutaneous or mucous manifestation. Bresgen, on the other hand, objects with reason I think, that cutaneous manifestations may sometimes be very tardy, appearing perhaps two years after the beginning of pituitary lupus, and for these reasons considers Schoeffer's growths as lupus tumors.

Olympitis, while placing Schoeffer's tumors in the tuberculous class, recognizes that they constitute a transition form between tuberculosis and lupus. Likewise his infiltrated form, "limited to lupus." Now, there is not only a relation between this form and lupus, but, as we shall see, an absolute identity.

Hinsberg[8] in a work on the relation of ocular affections to the nasal mucosa, reports four observations of pituitary tuberculosis and one of lupus.

I have soberly asked myself, upon reading this otherwise very interesting note, upon what characters the author depends to declare that one case is tuberculosis and another lupus.

Here, very much abbreviated, are two of his observations of nasal "tuberculosis:"

1. Young girl of 15. The right inferior meatus is filled with a granular mass, in places ulcerated, coming apparently from the inferior turbinate; on the left side a similar tumor but much smaller. In the pharynx, the right tonsil, the pillars and neighboring parts of the velum are infiltrated, thickened and pale. The surface is studded with small nodules but without ulceration; this condition extends to the epiglottis which is thickened, immobile and covered with similar nodules. Later on, there appeared nodules on the mucosa covering the arytenoids.

2. Girl of 17; came to the clinic because of an eruption on the lobule of the nose and left lower eyelid. There was found at the right nasal opening a severe eczematous condition with crusts; the skin at the summit of the nose was reddened, with some nodules. The right side of the septum, at its lower anterior portion, shows a mass of granulations covered with crusts. The right inferior turbinate is similarly affected, and the left is covered with crusts and granulations. In the nasopharynx, right side, there are some nodules; some nodules also on the skin of the left lower eyelid.

Here now is the case labeled lupus:

Man of 48, previously treated at the dermatologic clinic for cutaneous lupus of the nose. At the time of examination the nasal fossae show, besides a deviation of the septum, numerous small nodular tumors on the mucosa of the left inferior turbinate and both sides of septum. Three years afterward there was seen the same turbinate lesions with an added perforation of the cartilage.

Evidently in these three cases there was a single disease.

Massei, in his work published in the Revue hebdomadaire de Laryngologie,[9] on nasal tuberculosis, declares the division into lupus and tuberculosis to be superannuated, and proposes a complete erasure of the term lupus from the medical vocabulary.

Escat, on the other hand,[10] has never had occasion to see any other manifestation of tuberculosis in the nasal mucosa than lupus.

It is thus to be seen that the confusion is complete, and, as I said at the outset, the question of nasal tuberculosis is to be taken up anew. I believe that it will be of interest to examine the accepted classic forms of nasal tuberculosis, and I hope to be able to demonstrate that they may be reduced to a sole and single modality—lupus.

I will therefore take up successively the following questions:

1. Is there an ozenous form of nasal tuberculosis?

2. Is there ground for recognizing an osteo-cartilaginous form of nasal tuberculosis?

3. Is there ground for making a distinction between tuberculosis and lupus of the pituitary membrane.

After solving these questions, I will discuss the clinical aspect of nasal tuberculosis, its etiology, its progress, its complications, its diagnosis and its treatment.

I.—IS THERE AN OZENOUS FORM OF NASAL TUBERCULOSIS? RELATION OF OZENA AND TUBERCULOSIS OF THE NASAL MUCOSA.

Let us first examine the observations upon which this clinical form has been established. These cases, now ancient, are three in number, and I will give them in extenso. One belongs to Hajek, the other two to Demme.

(a) Hajek (in These Boutard). Tuberculous ulceration with ozena in a youth of 13.

Youth, aged 13, nose flattened, exhaling a very bad nasal odor. The interior of the nose was filled with crusts, the bony septum was partially destroyed, and there were pustular granulations in the bottom of the ulcers. The mucosa of the middle and lower turbinates on both sides was dry, thinned and greatly atrophied. The patient had been in this condition for a year and a half. For a year there had been considerable swelling of the cervical glands, which had improved under iodized baths without any change in the nose.

A minute examination did not show any symptoms of hereditary syphilis; his parents having died long before, no information in this respect could be obtained. For diagnostic purposes I removed a piece the size of a pea from the edge of the septal ulcer. I made a section and found in a spot rich with cells, characteristic tubercles, among which some had at their center giant cells with their nuclei and a few bacilli. Others appeared to be undergoing caseous degeneration. Application of lactic acid to the ulceration. His condition improved temporarily, but the ulceration finally increased progressively and the malady followed its usual course.

(b) Demme.[11] (Berl. klin. Woch., 1883, Bd. xx, No. 15): In a particularly grave case of rhinitis, which presented itself as an infantile scrofulous ozena, the writer observed in a 12 months old boy little groups of grayish yellow nodules showing up clearly on the nasal septum after cleaning out the crusts which obstructed the nose. These nests were irregular and had small ulcerations on their surface. The bottom of the ulcerations was filled with a yellow sanious mass covered with pus. In this case hereditary syphilis was determined with certainty. However, it turned out to be an ulceration of the mucosa, of a tuberculous nature. In fact, the child was seen a few months afterward, and he then had pulmonary phthisis."

(c) Demme (id.).

February 28, 1885, there was brought to me a little boy, 8 months old, Fritz B., who had ozena. A complete physical examination of the child led to the exclusion of hereditary syphilis. The antecedents were in accord with this view, and the diagnosis of hereditary tuberculosis was made. It was then learned that the child had been taken into a family in which the father had acute miliary tuberculosis. The ozena appeared in the baby at about his sixth month, two months after his residence with this family. On the right septal mucosa was seen a group of small nodules identical with those above described. Some of these were the seat of lenticular ulcerations of unequal depth and with crenated borders. In five preparations from the nasal discharge made February 28, only one contained tubercle bacilli. March 1, after a nasal irrigation with warm water, the patient had a violent attack of sneezing, followed by an abundant nasal secretion which when examined under the microscope after staining with fuchsin, revealed a considerable number of bacilli, almost as great as the number found in acute phthisis. March 2, there were convulsions with high fever; another bacteriologic examination with the same result. With the symptoms of an acute meningitis of rapid onset the patient died March 5, after frequently repeated general convulsions and contracture of the lower extremities.

At autopsy there was found a tuberculous meningitis of the base of the brain. Numerous miliary tubercles were along the branches of the Sylvian vein as well as the capillaries of the inferior surface and the frontal lobes, notably the peripheral cerebral veins, anterior and inferior. No tubercular products in the brain or of the cord. The lungs, bronchial glands and abdominal organs were exempt from tuberculosis. The mesenteric glands appeared swollen and soft. No caseous foci were found. In the axillary folds were some isolated hypertrophied glands; no caseation on section.

The ulcerous masses in the nose, when microscopically examined, showed bacilli in moderate numbers, either in isolated groups or included within giant cells.

In some sections from the miliary tubercules of the pia mater a very few bacilli were found. The author concluded that it was a case of direct tuberculosis infection of the nasal mucosa, coming directly from the foster father, who had pulmonary tuberculosis. The tubercular infection of the nasal mucosa was carried by the vascular route, and thus implanted in the pia mater and base of the brain.

As may be seen, these observations have dealt with a special form of ozena, and all three mention ulcerations of the mucosa, although everybody nowadays agrees upon the absence of ulceration in true atrophic rhinitis.

The ulceration mentioned in Hajek's case was of bone Now, in none of my personal observations of nasal tuberculosis nor in those which I have collected from various publications (covering a century) have I found this species of ulceration. It may be said then that perforation of the bony septum in nasal tuberculosis is a rarity.

On the contrary, it is a classic feature of tertiary syphilis of the nasal fossae which may be accompanied at an advanced period of evolution by atrophy of the inferior turbinates. So that one may ask if, in this case, there was not a tubercular infection engrafted upon a nasal syphilis. To be sure, the observation states expressly that there were no stigmata of hereditary syphilis, and the great authority of Hajek should be a guarantee of the fact. However, specific treatment does not appear to have been tried. This is plainly a valuable item which is missing.

The observations of Demme have dealt with an extremely rapid tuberculosis. In the case where a bacteriologic examination of the secretions was made, numerous bacilli were found, "almost equal in quantity to that found in acute phthisis." Autopsy showed numerous miliary tubercles along the branches of the Sylvian artery and vein. One gets the impression that it was a rapid, massive, recent infection, which might have been subsequent to the appearance of the ozena.

So, one might ask himself if, in these cases, there was not a simple coincidence, or rather a juxtaposition of acute tuberculous infection and true ozena. Hence these observations do not seem to me beyond criticism.

I may add that the rarity of these cases contrasts singularly with the considerable number of cases of pituitary tuberculosis not accompanied by ozena. Is it not astonishing if Koch's bacillus has ozenogenous power, not to see it oftener in action?

However, these cases ought to be taken into consideration. They show, indeed, one thing: that Koch's bacillus may live in good comradeship with the bacilli of ozena.

Clinical researches which I made long ago have convinced me that atrophic rhinitis is only a larval form of nasal tuberculosis. (Soc. Francaise de Laryngologie, June, 1907.) But Koch's bacillus does not produce ozena, the bad odor; it causes the atrophy, the sclerosis of the turbinates, just as it elsewhere produces renal sclerosis or periportal sclerosis, and this atrophy through the new condition which it provokes in the nasal fossa (drying of secretion, etc.), would favor secondary infection by the microorganisms of ozena.

Thus understood, tuberculous ozena would be relatively frequent. Its existence is not yet demonstrated. Further researches are necessary.

II.—IS THERE GROUND FOR RECOGNIZING AN OSTEO-CARTILAGINOUS FORM OF NASAL TUBERCULOSIS?

Osseous manifestations of nasal tuberculosis are extremely rare; I know of but two cases, which I shall recount:

Case I (Verneuil-Ruault in These Boutard). In 1887 Verneuil sent to Ruault a woman of 40 whom he had treated two years for a nasal affection which began as a tumor in the floor of the right nasal fossa. Verneuil did an ablation, and an examination by Nepveu showed tuberculosis.

A cure could not be obtained. The tumor did not recur, but the wound ulcerated and the cartilaginous septum was soon destroyed. Weekly cauterizations with the thermocautery for two years, when the patient was sent to Ruault.

The latter found a grayish ulcer with jagged edges occupying over a third of the floor in the right nasal fossa and a smaller area in the left; no trace of the cartilaginous septum except at its upper part. The vomer was carious in its whole anterior portion. At the opening of the right naris there were some small tubercles on the skin, of lupus appearance. For several weeks the voice had been roughened and deglutition painful.

Pulmonary and laryngeal lesions carried off the patient.

Case II (Koschier, Case XV in the work already cited). Woman, H. T., 22 years, admitted May 17, 1894. For a year had noticed that air did not pass through the nose.

Externally the nose presented on both sides a very large bridge, larger than normal. The skin was inflamed and red, especially on the bridge. Palpation of the nose caused lively pain. The wings of the nose were swollen somewhat, but of natural consistence. Both nasal fossae were filled with a large tumor causing complete impermeability of the nose, a soft. nonulcerated tumor seated on the septum in front and completely hiding the septum laterally and posteriorly. Nothing abnormal by posterior rhinoscopy. Throat, larynx and lungs normal; no bacilli in sputum in spite of repeated examinations. Examination of a portion of the tumor showed tuberculosis.

Under anesthesia the greater part of the tumor was removed with a bistoury; it was then found that the lateral parts of the nasal fossa, in particular the inferior turbinate and floor, were greatly infiltrated and showed ulcerations in several places. With the probe denuded bone was felt in several spots. Operation was then stopped; tamponment with iodoform gauze; subsequent dressing; application of lactic acid and insufflation of iodol. Death July 3, 1895. At autopsy, acute basilar tubercular meningitis with acute internal hydrocephalus and cerebral edema. Chronic tubercular periostitis and perichondritis of the nose. Tubercular otitis media and caries of the mastoid process. Chronic tuberculosis of both pulmonary apices.

Authors who have occupied themselves with nasal tuberculosis do not fail. for the most part, to quote the opinion of Volkmann upon primary caseous osteomyelitis of the nasal bones, invading the nasal mucosa secondarily, which he regards as a frequent cause of ozena. Now, when this work is consulted, it is not a little surprising to find that it deals with the character and significance of fungous arteritis. In one passage in the work, Volkmann voices the opinion above mentioned, but without the support of any clinical facts, much less any histologic examinations.

As for the cases above given, I find them extremely debatable.

Nothing proves that the bone lesions in the first case were tubercular. I even believe that weekly treatments with the thermocautery for two years were amply sufficient to provoke the osteitis of the vomer.

As for the second case, I hope not to be taxed with exaggeration in saying that if this observation had been presented to an unprejudiced reader, omitting the bacteriologic examinations, he would have diagnosticated tertiary nasal syphilis with osteo-periostitis of the nasal bones proper.

There is nothing lacking. Thickening of the nasal dorsum, with redness, edema, and lively pain upon pressure, and smooth swelling from infiltration of the inferior turbinate which the probe found to be denuded and carious at the bottom of an ulceration.

Trial with specific treatment is not mentioned, and it may be asked if it was not a mixed lesion, syphilitic and tubercular. The fact is not impossible.

At the Vienna Laryngologic Society, November 9, 1904, Dr. Marschik[14] reported a case of tuberculosis of the nose and velum resembling syphilis, and improved by mercurial treatment. In the same session, Dr. Heindle[15] stated that he had seen analogous cases where there was no trace of syphilis in the history, in which examination demonstrated the existence of tubercles and which healed under antisyphilitic treatment.

Hence, these cases are not convincing as to the solely tubercular nature of the bone lesions observed.

In any event they do not suffice to justify the existence of an osteo-cartilaginous form of nasal tuberculosis. I do not mean to deny the possibility of tuberculosis of the nasal bones; here, as in other bones, Koch's bacillus could propagate, but that is a thing altogether different from what we are concerned with in this paper. And I do not believe that the question of nasal tuberculosis is clarified by including tubercular osteitis of the maxilla, frontal or ethmoid.

It will be seen in the chapter on pathogenesis that I completely reject, on clinical grounds, the osseous origin of common nasal tuberculosis.

III.—IS THERE REASON TO MAKE A DISTINCTION BETWEEN PITUITARY TUBERCULOSIS AND LUPUS?

We have seen, at the beginning of this paper, how much confusion reigns in authors' descriptions, and what one calls common tuberculosis another calls lupus.

To solve the question it will then be necessary to set forth as precisely as possible the characteristics of pituitary lupus, which have been only vaguely described in classic treatises. These characteristics being known, we can review the cases reported as nasal tuberculosis, and see if the lesions therein described differ or not from those considered to be lupus.

I will first take up the characteristics of lupus and discuss the cases afterward.

A.—Objective Signs of Pituitary Lupus.

The objective symptoms which I am about to describe are those which I observed in twenty patients who were attacked simultaneously with lupus of the nose or face and with pituitary lesions, most of whom were sent to me by Dr. Balzer. Four only had intranasal lesions alone, in every respect similar to those observed in the ones who had cutaneous lupus.

In spite of the apparent identity, I subjected two of these patients (Cases IV and XV) to mixed antisyphilitic treatment. The treatment was absolutely without effect upon the lesions. To make it stronger, I inoculated two guinea-pigs with a bit of mucosa taken from each of these patients; both inoculations were positive.

The failure of specific treatment and the positive inoculations satisfied me of the tubercular nature of the lesions. The coincidence of cutaneous lupus showed, in another way, the true significance of these lesions.

It follows from fifteen cases published herewith further on (to which can be added six others published in the Annals of September, 1906[16] and of January, 1906)* pituitary lupus may assume four different forms or aspects:

Nodular lupus,
Vegetating lupus,
Lupus tumor.
Ulcerous lupus.

1. *Nodular lupus.*—It is characterized by the appearance on the pituitary mucosa of nodules, usually of a pale rose color, The number varies according to the time when seen; there may be only one or two at the beginning. They are two or three times the size of a pin-head. They lack the lustre of normal mucosa, being dull and roughened.. Soft upon con-

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*The two latter cases I published as naso-lacrymal tuberculosis. They were, in fact, lupus, for the patient in the second case, Louise B., had a double facial lupus, and I have recently seen the little patient in Case 1, who showed typical pituitary lupus.

tact with the probe, they are separated from each other by small irregular grooves, some ulcerated, others cicatricial.

When they are numerous they infiltrate the mucosa in a series of nipple-like lobules separated from each other by the grooves above mentioned—this is the mammillated infiltration characteristic of pituitary lupus. At this stage the mucosa is soft to the probe and does not contract under the influence of vaso-constrictor medicaments.

The more or less abundant crusts often mask lesions to which they slightly adhere. The lesion may be seated in any part of the nasal fossae, but it always begins anteriorly, as we shall see in the chapter on the course of the disease; for the moment I want only to give the characteristics of lupus in a fashion such that they may be compared with those in case reports which, rarely it is true, have dealt with lesions at their commencement. The mammilla with grooves surrounding it is, in a way, the elementary lesion of pituitary lupus—it is always found in some form.

2. *Vegetating Lupus.*—It is characterized by a more or less extensive hyperplasia of the mucosa—a pale rose or bluish lilac hyperplasia with a series of crests separated by little ditches.

I cannot make a better comparison of the appearance given by the mucosa than to the geographic wall charts in relief designed for primary classes. One sees minute hillocks separated by miniature valleys. Each of the hillocks, sometimes rounded like a dome, sometimes terminating in a sharp crest, is made up (the same as the valleys) of an agglomeration of very small nodules the size of a pin-head, separated by extremely narrow grooves, giving to the lesion as a whole a muriform aspect which is peculiar to it.

These lesions always feel soft to the exploring probe. Especially upon curetting is this extreme softness perceptible. The softness is even a good means of diagnosis when, in the course of treating lupus, one is uncertain whether a given region is or is not lupic.

The group of lesions occupies a more or less considerable surface, according to the period when observed; at the beginning it may occupy solely the anterior zones of the nasal fossae (Case IV), or a large part of the septal mucosa (Case III); later it may involve almost all the nasal mucosa (Case VI).

3. *Lupus Tumor.*—This merits description apart from the

vegetating form. Pedunculated or sessile, of variable size (that of a hazelnut, a walnut, the entire nasal fossa in one case) the tumor is of a pale bluish white color, sometimes red (probably in consequence of secondary infection). Of smooth appearance on superficial examination, one sees on its surface (personal case) a noteworthy number of grayish yellow elevations the size of a millet seed. It is elastic and bleeds little or not at contact with the probe.

It may be almost anywhere in the nasal fossa; on the septum, floor, inferior turbinate, middle turbinate (anterior or posterior), as may be verified by reading my cases.

Finally, it is almost always accompanied by some other manifestation of pituitary lupus (nodules, vegetations, perforation of the cartilaginous septum). I may add that it may be single or accompanied by other identical manifestations; we then have a form intermediate between the vegetating form and tumor.

4. *Ulcerating Lupus.*—Ulceration is an eventuality which may ensue in the course of the preceding varieties. It is an ulceration with irregular borders, sometimes polycyclic, with granular bottom, sometimes of a necrotic appearance. Resulting from the breaking down of preexisting lupic tissue, it is always surrounded by a zone of mucosa showing one of the lesions above described. Most often it occupies the cartilaginous septum, but it may be seated upon the subseptum, the floor, or the inferior turbinate.

If it comes on in the course of a vegetating lupus, it is always found to be elevated above the healthy mucosa; it thus gives the geographic relief chart appearance, which I have mentioned. It only attacks the cartilages late.

If it develops upon a nodular lupus, it easily causes perforation of the septum, which is the more easily produced in bilateral lesions of the septum. It is probable that the loss of circulation in the cartilage from destruction of its nutrient mucosa, brings on the necrosis.

The perforation presents peculiar characters: It is rounded and regular. The dimensions vary from a pea to a two franc piece. The border is surrounded with a collarette of fungosities, soft, bleeding, pale or red, more or less voluminous.

It is not rare—a somewhat characteristic fact—to find a part of the periphery cicatricized and showing a thin border, while the rest, in full swing, is thick, tripled in volume and fungous.

A capital fact: it attacks exclusively the cartilaginous septum; I have never seen lupus accompanied by a bone lesion.

Perforation of the cartilage is an extremely frequent manifestation of lupus at a certain period of its evolution, hence it has high diagnostic value. Its presence, accompanied by other lesions difficult to interpret, not involving the bone, is sufficient, I hold, to give these lesions a lupic significance.

Ulceration, speaking precisely, is less a form than an end-stage of lupus.

I would say the same of the stenosing form, which Raulin describes in his thesis. This is an atresia of the naris, encroaching more or less upon the nasal fossa, and simply means the cure of an old lupus of the vestibule and anterior nasal fossa. It is strongly indicative of lupus.

Sometimes it is characterized by the formation near the posterior narinal border of a vertical and frontal thickening with an upper concave edge, three or four millimeters high, barring in a fashion access to the nasal floor. I have seen this in one case; it has also been pointed out by Mygind[37].

In other cases there is actually produced a fibrous tunnel through which the parts constituting the nasal fossa are seen with difficulty (Raulin and Cases X, XI, etc.).

I wish finally to call attention to three frequent phenomena in the course of pituitary lupus, and consequently of great significance if we should find them mentioned in case reports. They are: Destruction of the alae nasi, lymphangitis of the lobule and "eczema" of the nares.

The nares may be more or less destroyed, eaten away by the breaking down of lupus cutaneous nodules. Various deformities result, the least of which is more or less reduction of the narinal dimensions with characteristic vermicular cutaneous cicatrices, or if extensive, the horrible disfiguration called the "death's head." This is not the destruction which I have in view, but the progressive destruction of the alae nasi, unilateral or bilateral, without any nodules in this region. I have noticed it several times, especially in patient of Case I. The alae nasi were the seat of a smooth scar tissue production which led to their progressive wasting; during the five years that I cared for this patient I have seen them become an arch, the concavity below, thus gradually uncovering the septum. In another case I have seen almost total destruction of the left ala, exactly resembling cases of rhinelcosis. There may have been a tuberculous trophic process in this case, or in the first, a sclerodermic tuberculosis, analogous to that recently described by Milian[17].

I consider the symptoms in both cases sufficiently characteristic of lupus. Lymphangitis of the lobule is very frequent in the course of pituitary lupus. It gives to the extremity of the nose, a swollen, red aspect, which I have often noted, sometimes long before the appearance of the cutaneous nodules, at times even the initial symptom at the beginning of pituitary lupus (I shall recur to this fact). Is it a tuberculous manifestation or simply a transitory infection of the skin with other microorganisms? I cannot say; it is frequent, may be slight or intermittent, and seems to me to have much significance in being always associated with pituitary lupus.

I would say as much for eczema of the nares, which I have frequently found mentioned in observations of pituitary tuberculosis. This eczema may well be a tuberculous manifestation.

Hutchinson (cited by Lenglet, *Pratique Dermatologique*, p. 323) admits that eczema may precede the evolution of lupus, but he remarks that some cases of lupus preserve, during their whole course, the aspect of prelupic eczemas. This is also the opinion of Jodassohn (id.), who regards certain chronic eczemas of the nose and upper lip as the preliminary stage of lupus. Tuberculin often gives a positive reaction in these cases. Also, we must distrust certain varieties of so-called chronic eczema which transform themselves into lupus. Certain prelupic eruptions are tuberculous, and some eczemas are doubtless of the same origin and merit the appellation of tuberculous eczemas, or rather prelupic eczematiform dermatitis (Lenglet, loc. cit.).

When we recall that the progress of lupus is extremely slow; that it lasts for years; that recurrences are extremely frequent in spite of treatment methodically applied, we possess those principles which permit us to criticise case reports.

B.—Criticism of Case Reports.

From the above exposition, based upon my twenty observations, I believe I have a right to draw the following conclusions:

1. We can surely list as pituitary lupus the cases of tubercu-

losis where there was remarked one or several of the following signs:

Mammillated infiltration of the mucosa, which I regard as the typical lesion of lupus.

Perforation of the septal cartilage;

Destruction of the alae nasi.

Coexistence of other lupic lesions of nose, cheek and face in general.

Coexistence of other lupic lesions of the mucosa.

Narinal atresia in tunnel form.

2. We can almost, with certainty, include as lupus the cases where one of the following signs is shown:

Long duration of the affection.

Frequent recrudescences in spite of methodical treatment.

Lymphangitis of the lobule.

The presence of vermicular cicatrices on the lobule or at the narinal orifices.

The presence of "eczema" of the nares, which, in several cases, seems to me to have been nothing else than vestibular lupus.

These signs acquire the value of certainty if they coexist with one of the symptoms above mentioned.

The absolute indolence of lupus contrasts with the pain, often sharp, of miliary tubercular ulcerations.

I may add that the rarity or absence of bacilli upon histologic examination is also in favor of lupus.

Let us now examine, in the light of these facts, the cases enumerated in my table.

If we throw out nine case reports which are really too brief to be utilized (Wroblewski I and II, Chiari III, Capart, Silberstein I and II, Herzog V, VI, VII) we see that the cases can be analyzed as follows:

Tumors	44
Ulcerations	20
Infiltrations	21
Mixed lesions	2

The tumors comprise 29 certain lupus tumors and 1 probably lupus. Only 14 of these cases (which includes Schaeffer's 6) are noted as the only pituitary lesion. I do not hesitate to consider them as lupic tumors; in fact, nothing distinguishes them from other tumors accompanying pituitary lesions; like them, their structure never or rarely revealed bacilli, and like them, were in patients clear of pulmonary lesions; in a word, nothing authorizes their separation.

Of the twenty ulcerations, eighteen appeared as the ultimate manifestation in pulmonary tuberculosis. Their aspect did not differ at all from granular lesions met with upon any other mucosa (ulcer with scooped out borders, very painful, miliary granulations in a collarette about the periphery) and bacteriologic examination showed the presence of numerous tubercle bacilli. We may conclude that they were terminal granular ulcerations in cases of pulmonary tuberculosis. Two other cases, however (Baurowicz, Michelson II) are manifestly cases of lupus.

In the twenty-one observations with infiltration of the mucosa, only four (Cases III and IV of Hahn, VI of Koschier, and IV of Herzog) are probably lupus; all the others, seventeen, are absolutely lupus. I consider as mixed lesions, granular and lupic, the lesions described in case XVI of Koschier, I of Bar, and the case of Tournwaldt.

The muriform hyperplasia and tumor of the inferior turbinate in Tournwaldt's case, the perforation of the septum in Koschier's case, the smooth pseudo-polyp masses in Bar's case are lupus manifestations as all the other observations testify. They contrast singularly in their slow progress, indolence and absence of ulceration, with the pain of other lesions, the granular ones of the pharynx, larynx and floor of the nasal fossa.

Lupus is slow in evolution, insidious and goes unnoticed until it becomes obstructive. One treats an eruption or a granular ulceration in the pharynx and larynx; in examining the nasal fossae lesions are found which one believes to be contemporaneous and of the same character, but they are of an earlier date and a different sort.*

We see then, to sum up, that there exist only two varieties of nasal tuberculosis: Miliary tuberculosis with frank and unequivocal characteristics, and lupus which comprises: tubercular tumors and the majority of tubercular vegetations. I say the majority, for certain infective granular ulcerations may bud and give rise to vegetations as in the case reported by Band and Tixier, but the granular type of ulcer remains easily recognizable.

The facts seem to me clearly demonstrated: Under the names of tuberculous tumor, vegetating tuberculosis and lupus, writers have described a sole and single thing. This identical thing should be called lupus, for it is characterized objectively and functionally by the same symptoms which, as I have shown already, characterize pituitary manifestations of those having skin lupus.

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*I could cite other examples of manifestations described as tuberculosis which are really lupus.

Kayser (*Monats. f. Ohrenheilkunde,* April, 1901), cites a case of primary nasal tuberculosis in which he described, besides perforation of the cartilaginous septum, the mammillated infiltration characteristic of lupus.

Symond describes under the name of tuberculous ulceration of the septum (London lar. Society, 1897, in *Rev. hebdomadaire,* 1897), a plain case of lupus.

Williams (London lar. Society, April, 1897, in *Rev. hebdomadaire,* 1897, 2, p. 899), describes as common tuberculosis a tumor coinciding with lupic pituitary manifestations.

This study is solely clinical. I will say at once that pathologic anatomy no longer permits a differentiation of lupus from tuberculosis of the nasal mucosa. M. Schmidt (*Die Krankheiten der Oberen Luftwege,* 3rd edit., 1903), after Massei, affirms that tuberculous tumors cannot be differentiated macroscopically or microscopically from lupic new formations.

IV.—ETIOLOGY.

Thus defined, pituitary tuberculosis ceases to be the clinical rarity taught in the classics. If cases of pituitary tuberculosis are relatively rare the number of lupus cases is, on the other hand, relatively considerable. One has only to read, to get an idea, the reports of various scientific bodies and especially the London Laryngological Society. I am even convinced that it is greater still than indicated by the number of lupus cases published. For one thing, lupus is painless, only slightly attracts the patient's attention and may heal of itself; some do not even ask medical attention. For another, very scattered lesions pass unperceived at the beginning.

My statistics confirm the classic figures relative to sex and age of predilection. There is a marked predilection for the female sex, which covers three-fourths of the cases in my list. The ratio is larger still in my personal cases, as I have had eighteen women to two men (90 per cent).

As to age, lupus attacks especially in middle life. By referring to my cases and tables, it will be found that the maximum of frequency was between 15 and 25 years, 44 per cent. There was equal frequency between 5 and 15 and between 25 and 50. Beyond 50 it is very rare. I have had one case at 63 and another at 71.

It always develops upon scrofulous soil. It often succeeds a strumous pseudo-atrophic coryza (Moure).

I believe, moreover, and I have tried to prove that pseudo-atrophic coryza, like atrophic rhinitis (with or without ozena) are larval forms of tuberculosis.

V.—PATHOLOGY.

To the two varieties, granular and lupic, belong two different pathologies.

Lupus is a local tuberculosis, primary, independent of pulmonary lesions. Miliary tuberculosis, on the contrary, is secondary to pulmonary lesions. In lupus the infection is direct; it is brought about by inhalation or direct carriage by soiled fingers. In miliary tuberculosis, on the contrary, the infection is carried from the lungs to the nasal fossae by the blood.

1. *Pituitary lupus is primary.*—All writers upon this subject are not agreed as to this.

Herzog, for example, in eighty cases of nasal tuberculosis only found twenty (one-fourth) where there was not pulmonary tuberculosis at the same time. All his cases of secondary tuberculosis belonged to the ulcerous form. My statistics, made up in part from the same sources as his, show differently.

Francis Steward[18] collated 100 cases of nasal tuberculosis, of which six were personal. He found that the pituitary lesions were primary in fifty-eight, secondary in thirty-seven, and doubtful in five. But he admits that the tuberculous tumor (which he distinguishes from lupus) is most often primary (33 out of 40).

Pistre (tuberculous pseudo-polyps) considers tuberculous tumors to be "most often secondary." This assertion is the more surprising from that author, since in his thirteen cases eight were manifestly primary tumors, three made no mention of pulmonary conditions and only two show the coexistence of tuberculous tumor and pulmonary lesion.

Pasch,[36] while recognizing that the tuberculoma (which he also distinguishes from lupus) is most often primary, has also seen it coincide with tuberculous infiltration of the lungs.

All my personal cases were primary lupus of the pituitary membrane. In no case (my personal statistics include twenty-two cases—sixteen new cases, four published in this journal in January, 1906, and two in November, 1906), did I find a pulmonary lesion.

In the cases collated from the literature, only nine coincided with tuberculous lung lesions (case of Tournwaldt, VI of Hahn, IV of Michelson, IV, VII, XI, XVI of Koschier, I of Hinsberg, and I of Bar).

Now the coincidence of lung lesion and pituitary lupus does not prove that the pulmonary trouble caused it. In none of these cases is the chronology recorded, and nothing permits the affirmation that the lung lesion preceded the nasal lesion. On the contrary, important reasons could be advanced in favor of a pituitary beginning.

We know that lupus makes extremely slow progress (see further on) which is figured by years.

We know, further, that its march is extremely insidious, and consequently that it may have long existed, when it comes the first time for examination. Now all these nine cases report, with slight pulmonary lesions, advanced lesions in the pituitary.

The patient of Tournwaldt presented pulmonary induration, but his nasal fossae were the seat of a muco-purulent discharge since infancy, and the lupus was quite extensive.

The patient in Case VI of Hahn had signs of softening and pulmonary shrinking, but the septal perforation testifies to the duration of the pituitary lesion. The patient in case VI of mine reported January, 1906, had only in 1902 a slight mammillation, the size of a pea in the inferior meatus which would have passed unnoticed if my attention had not been called to it; five and a half years later she had lesions of the cartilaginous septum and anterior thirds of both inferior turbinates, without perforation of the cartilage. No pulmonary lesion.

In Case IV of Michelson it is expressly noted that the patient dated his nasal obstruction several years back.

The same in Cases VII and XVI.

On the one hand slight lung lesions; on the other, advanced pituitary lesions. Logic holds that the latter began first. Moreover, these cases constitute an insignificant minority.

In fact, if we deduct twenty-one cases from the table where the pulmonary condition was not shown, we find thirty-seven cases of primary lupus to which must be added my sixteen personal cases and the six others published in January and December, 1906, so that there are fifty-nine cases of primary lupus against nine of lupus with pulmonary lesions. The primary lupus cases make, then, 90.5 per cent of the whole.

Another consideration, indirect but important, confirms me in my opinion. As Francis Steward[18] has remarked, if we add together the tuberculous lung cases of Willigh[19] and Weichselbaum,[20] we get a total of three cases of pituitary tuberculosis in 640 lung cases, that is, 0.46 per cent.

This shows that in coincidence of pulmonary and pituitary tuberculosis it is the nasal tuberculosis which came first.

It is not rash to think that the twenty-one cases in my table who had no mention of the pulmonary condition were primary pituitary lesions.

2. *Granular tuberculosis is, on the contrary, secondary to a pulmonary lesion.*—This appears from an examination of the cases reported by my colleagues in this essay. All the granular ulcerations therein recorded appear to me to be a terminal lesion in the course of pulmonary phthisis. There is but one case of granular ulceration of the pituitary membrane without a pulmonary lesion; this is the one described by Melzi[21] in the *Archives Internationales de Laryngologie*, a patient who came to him for a very pronounced nasal obstruction. The author made out a common hypertrophic rhinitis and did a turbinotomy. To his great astonishment he found on the meatal aspect of the inferior turbinate an ulceration comparable to ulcerations on the tongue. The borders were undermined. On the periphery were several characteristic gray granulations. The patient was young and clinically free from pulmonary tuberculosis. The ulceration was found histologically to be tuberculous but no Koch's bacilli were found.

This case is unique to my knowledge. It does not contradict the rule that a granular ulceration is most often a terminal phenomenon in the evolution of a pulmonary phthisis.

It is a blood infection. There is a bacillary discharge into the circulatory system from the pulmonary parenchyma which becomes colonized in the nasal fossa. Usually there is a massive baciliary discharge. One finds in these ulcerations numerous Koch's bacilli, contrariwise to what is found in lupus lesions where the bacilli are rare or undiscoverable. To sum up; there exist two modes of pituitary infection: the blood route producing miliary tuberculosis; the air route causing the appearance of lupus.

Escat supposed in the latter event, that the pituitary mucus had the power in some way of attenuating Koch's bacilli, modifying their virulence. The nasal fossae would thus be a sort of physiologic laboratory for the attenuation of bacilli.

VI.—SYMPTOMATOLOGY.

I have had especially in view in this study primary tuberculosis, lupus of the pituitary. I ought to say, however, a few words about granular tuberculosis.

A.—Miliary Granular Tuberculosis.

This presents in two forms: gray granulation, and ulceration. Gray granulation is extremely rare in the pituitary mucosa. It appears in the form of an eruption of small granulations, the size of a millet seed, gray, separated from each other by areas of healthy mucosa. They are usually seated upon the lower turbinate. If a swab culture be colored and examined a considerable quantity of tubercle bacilli will be seen.

I have found but two cases: One in the thesis of Olympitis which belonged to Millard, and another in Boutard's thesis which belonged to Hajek.

In the latter case the gray, translucent granulations were seated upon the inferior turbinate; a slide examination showed numerous tubercle bacilli. They went on very rapidly to ulceration. This rapid transformation is the rule, and doubtless explains the infrequency of a pituitary miliary eruption.

Granular ulceration shows nothing peculiar. It has the usual aspect of granular ulcerations as seen upon the tongue (undermined edges, collarette of miliary tubercles). It has a marked predilection for the anterior portion of the nasal fossae, the anterior part of the septum and the neighboring part of the upper lip. It is an ulceration almost as much narinal as nasal, and this perhaps explains the sharp pain accompanying this lesion as opposed to the remarkable indolence of lupus.

Granular ulceration of the pituitary mucosa has, then, a real existence, and, by its location, its character, the pain which it produces, the numerous bacilli in the scrapings, the coexistence of pulmonary phthisis, clearly distinguishes itself from lupic ulceration. It may, especially if infected, become vegetant, but it rarely goes on to perforation of the cartilage.

B.—Lupus.

I have given, at the beginning of this paper, the clinical picture of lupus at its height. There remains something to be said of its mode of onset, its functional symptoms, its course, and the condition of the facial lymphatic system.

1. *Mode of onset. The pathognomonic signs of pituitary lupus.*—I have already indicated the modes of onset in my work of January, 1906. Repeating what I have said there are:

A form of lupus with a lacrymal beginning. A form with a lobular lymphatic beginning. An impetiginous vestibular form.

I refer to this work for the details of these modes of commencement. I will merely say that these modes appear to me to have much practical importance. They deal with the external phenomena immediately visible to every practitioner, not requiring the special manipulations of the rhinologist. They are the revelatory symptoms of pituitary lupus. If the practitioner knows that lacrymation, or a rebellious impetigo of the vestibule, or a torpid and recurring lymphangitis of the lobule may be the index of a baffling nasal tuberculosis, he will have him examined by a rhinologist without delay for the maladies coming under this category, and thus have his patient avoid a long lack of treatment which might result in a lacrymal fistula, destruction of the lobule or the subseptum, ending in the horrible nose destruction called the "death's head" or "parrot beak."

2. *Functional symptoms.*—These are almost nil unless the lesion becomes obstructive. It is even probable that it may remain latent for a long time.

Then there appears a dryness in the nose, or rather a drying of the secretions in the form of crusts which are sometimes sanguinolent. No pain, neither spontaneous nor upon pressure. Then the lesion becomes vegetating, or, if the crusts are abundant, nasal obstruction begins. Little or no reaction upon the general health. Sometimes the patients look really florid.

3. *State of the facial lymphatic system.*—I have already dwelt enough upon the local symptoms. I wish to say only a few words as to the lymphatics of the face.

It is known that the pituitary lymphatics (Andre, "Les lymphatiques des fosses nasales," Th., Paris, 1905) follow two currents of opposite direction.

The lymphatics of the mid-posterior portion are directed backward and terminate by a superior trunk at the retropharyngeal ganglia and upper ganglia of the jugular chain; by a middle trunk at the subdigastric and jugular ganglia (the former of which also receives the lymphatics of the anterior route); and by an inferior trunk at the ganglia of the jugular chain near the bifurcation of the carotid.

The lymphatics of the anterior third of the pituitary membrane participate in the distribution of the nasal integument. They therefore present three collecting groups; a middle group which ends at the submaxillary ganglia, following the path of the facial vessels; a superior group which ends at the preauricular and parotid ganglia; and an inferior group to the sub-mental ganglia. Along the facial chain are found a number of very small ganglia, seen in twenty out of thirty-two cases by Poincetreau, who pointed them out; the inferior, situated upon the horizontal branch of the inferior maxilla, in front of the masseter; the middle on the external aspect of the buccinator; and the superior, comprising a naso-genial and a suborbital ganglion.

Theoretically, gland infection ought then to be systematic as follows: To a lesion in the posterior two-thirds of the mucosa would correspond a retropharyngeal or retrodigastric adenitis; to a lesion of the anterior part would correspond an inflammation of one or more face ganglia.

Retropharyngeal adenitis has not been reported and I have not seen it; but a slight adenitis could go unnoticed by reason of the difficulty of examining this region. Moreover, at the beginning the lesions are confined to the anterior part of the pituitary membrane. All the ganglion territories of the face have, on the contrary, been involved.

Personally I have noted: Submaxillary adenitis (Cases IV, V and VI); subdigastric adenitis (Case VII); cervical and submental adenitis (Case VIII); submaxillary and carotid (Case X); submaxillary, submental and premasseteric (Case XV); premasseteric (Case XVI); submaxillary and carotid (Case I, *Memoirs* of September, 1906).

I have noted in the histories which I have looked through: Suppurative preauricular adenopathy, with subangulo-maxillary adenitis (Case XII of Pistre); submaxillary adenitis (Herzog, III and IV; Chiari, II; Silberstein, I and II); submaxillary and cervical adenitis (Pistre, XI; Hahn, II and VI); adenitis of the cervical glands (Hinsberg, IV). I recall also the two cases of Gelle and Poulard (Th. of Poulard[28]).

I have seen along with a unilateral nasal lesion bilateral adenitis, a fact which anatomy well explains (crossed distribution of lymphatics).

Apparently not much attention has been given to the condition of the lymphatic system in nasal tuberculosis. In eighty cases which I have collated I have found adenopathies in only ten, usually submaxillary. Andre in the clinical part of his thesis notes only the case of Poulard, previously mentioned. Gelle in his work[38] on adenopathies of nasal origin, remarks that writers are mute upon this question not only as to tuberculosis but also as to lupus, most authors making this distinction However, it appears to me to be frequent, for I have found it in ten of twenty-two cases, or 45 per cent. I believe it is even more common than the figures indicate, for at the beginning, I did not systematically explore the regions where I might find adenitis.

This examination of the ganglia has some diagnostic import, for, if along with adenitis there is no skin lesion, one will be led to examine the nasal fossae and perhaps find lupus lesions at their beginning.

Above all, as we shall see, it has pathologic importance. As the pituitary lymphatics follow the course of the tegumentary lymphatics, one may ask if the adenopathies which I have found with relative frequency, as remarked, are not due to skin lesions which sometimes coexist with pituitary lesions at the time when the patient comes for examination.

There is nothing in this. For if in a small number of cases the adenopathy does coincide with the lesions of the nasal integument (as in Cases V and X and Case IV of January, 1906), nothing goes to show that the adenopathy did not precede the cutaneous lesion; and if, in others, an interpretation of the adenitis is complex because of concomitant lesions (Case II, September, 1906), in the very large majority of cases (seven out of ten) the adenitis appears in the course of a primary pituitary lupus. Facial adenopathies, therefore, are evidence of the pituitary lesion and should cause it to be sought for.

4. *Progress, duration, termination, prognosis.*—The progress is extremely slow, torpid and insidious. It may exist six years (Case VII), eight years (XII), or ten years (V) without revealing itself except by insignificant symptoms, or simply by lacrymation. This slowness of evolution may be still greater, as witnesses Case X of Koschier. This was a woman of 71, who, together with a septal perforation, had some other small pituitary lesions. Twenty years previously she had been treated for a septal neoplasm; for twenty years then the lupus had remained limited to the anterior part of the nasal fossae, without having progressed.

Hence the duration of pituitary lupus is to be measured by years. Sometimes, however, probably as a result of secondary infections, it takes a more rapid course. I allude to those cases of lupus vorax, which in a few weeks destroy the cartilaginous septum, the subseptum, and the lobule of the nose; the diagnosis from syphilis may give difficulty in such cases. An important feature in the course of lupus consists in frequent recurrence as all cases show.

Lupus has also some tendency to spontaneous cure by reason of fibrous tissue production in the lesion which here, as everywhere else, tends to smother the lesions which it surrounds. It is true that, while it often heals in one place it breaks out afresh in another, in the cavum or in the larynx as we shall see. However, the formation of this fibrous tissue, the presence of which Garel and Collet[89] used in distinguishing histologically lupus from active pituitary tuberculosis, has the result of bringing on an atrophy of the inferior turbinates; and in an advanced lupus (especially if the lesions predominate in the inferior turbinates) it is common to see the nasal fossae considerably enlarged by a sort of lupus atrophic rhinitis, a novel proof of the role of tubercular toxins in producing turbinal atrophy. But if lupus does heal sometimes spontaneously it sometimes resists the best and most persistent treatment.

In a certain number of cases the lupus patient does not get well and dies of pulmonary tuberculosis. I have seen this eventuality once. Thus, while the patient with pulmonary tuberculosis never, so to speak, becomes lupic, the reverse is far from rare (Lenglet).

All these conditions show us that nothing is more difficult than to predict the future of a pituitary lupus, and fully justifies these words of Massei: Fears and hopes make up the prognosis; hopes in view of the benignity and slowness of the malady, permitting radical intervention; fears because of the complications which may involve other organs. Sometimes, after several years of reasonable hope, there come localizations in the lung, the larynx or the meninges, with a rapid course, carrying off the patient who was considered cured.

VII.—COMPLICATIONS.

If we can compare the nasal fossae to a laboratory where Koch's bacilli are attenuated, we must also recognize that this same laboratory, after attenuating them, spreads them in every direction. Situated right at the center of the face, at the beginning or end of many organs, it is not astonishing for the nasal fossae to scatter bacilli into these different organs.

Forward we see the pituitary membrane continuous by insensible transition with the skin of the face. In the inferior meatus it is continuous with the lacrymo-nasal canal; in the middle and superior meatus with the various sinuses of the face.

Behind, it continues, the cavum being intermediary, into the mucosa of the Eustachian tube and the tympanic cavity, and of the pharynx, esophagus and larynx.

Having so many paths which the bacilli may follow up, we have so many varieties of complications to study.

A.—Cutaneous Complications. Relations of Pituitary and Facial Lupus.

At first no account was taken of pituitary lesions in producing cutaneous lupus. The pituitary lesions were considered to be due to the spread of skin lesions to the nasal mucosa. Then it was recognized that lupus of the nasal mucosa could appear without any preliminary skin lesion. Primary lupus had acquired citizen's rights (*Th. of Raulin,* 1889). In spite of this, probably on account of the dominance of old notions, nobody saw that lupus of the mucosa could spread to the skin. Raulin himself believes that facial lupus is usually a primary skin infection, while, exceptionally, it is caused by a pituitary lesion.

The more and more frequently established coincidence of pituitary lesions with facial lupus forced a study of their relations, and it was asked if, instead of being an epiphenomenon in the course of facial lupus, the pituitary lesion was not the source of the cutaneous infection, and consequently the essential thing.

Rhinologists and dermatologists defend both opinions. The classic authorities believe rather in a primary cutaneous infection.

Lupus of the nasal fossae, says Lermoyez (*Therapeutique des maladies des fosses nasales*) may be: (a) primary when it begins in the pituitary membrane; (b) secondary, when, which is ordinarily the case, it first attacks the skin of the face and subsequently enters the nasal fossae.

Garel (*Diagnostic et traitment des maladies du nez,* 2nd edit.) after first believing in the rarity of primary pituitary lupus, now considers it relatively common, but, besides lupus of the nasal integument by spread from pituitary lupus, he believes in a primary lupus of the nasal integument whose nodules are much more destructive than the nodules of the former variety.

Menier[24] in his excellent manual thinks that most often when cutaneous and mucous lesions coincide, the former appeared first. On the other hand, Chiari[25] often remarks in the anamnesis of numerous cases of facial lupus, that these individuals, long before the appearance of the lupus, had a sore at the nasal entrance.

Bresgen[30] believes, as Neisser did before him, that facial lupus, as a general rule, is only an extension of a mucosal lupus.

Escat (cited above) thinks that cutaneous lupus is always consecutive to a pituitary lesion.

Molinie[26] believes that primary pituitary lupus is relatively frequent.

Audry[27] thinks that "skin lupus is usually if not always secondary to initial tubercular foci situated upon neighboring mucosa or deep tissues."

Sticker[29] professes the same opinion. Meneau and Freche[28] found in twenty-five cases of facial lupus, twenty-one where the nasal mucosa was the prime cause. There had been no rhinologic examination, the authors basing their opinion on the patient's nasal history (hypersecretion, crusts). They are convinced that "the frequency of lupus with a pituitary beginning, would be still greater, contrary to general opinion, if the nose were examined systematically and if nasal lupus could be detected at the outset." At the same time they do not consider lupus at the center of the cheek as being consecutive to pituitary lupus.

Lenglet (*Pratique dermatologique*) admits two methods of producing cutaneous lesions: (1) Cutaneous inoculation (kissing, scratching, former dermatoses). (2) Propagation from a lesion in the nasal mucosa. He seems to believe, with Raulin, that primary cutaneous lupus is the most frequent mode. Like Meneau and Freche he does not link lupus of the face, other than the nose, with pituitary lupus.

Professor Gaucher in his *Traite de dermatologie* is eclectic; various modes of pathogenesis of facial lupus are possible.

As for myself, I am firmly convinced of the pituitary origin of lupus of the nose and face.

This opinion on the one hand is based upon case reports which I consider unassailable; and on the other hand is the only way of explaining all the topographic modalities of facial lupus.

If certain dermatologists do not admit the pituitary portal of entry, it is because they do not understand very well the bond of union between the nasal lesion and the cutaneous nodule. "If it is right to regard lupus of the alae nasi as the result of a mucosal lupus, it is less evident that there is always a relation between lupus in other facial regions and lesions of the neighboring mucosa." (Lenglet, *loc. cit.*)

This bond of union is the lymphatic system.

We have now to study the case reports upon which I base my opinion, and the role of the lymphatics in the appearance of facial lupus.

1. *Facts which demonstrate the pituitary origin of nasal and facial lupus.*—My twenty-two observations of pituitary lupus may be divided into three classes: (a) Pituitary lupus without cutaneous lesion; (b) cutaneous lupus coinciding with pituitary lupus; (c) cutaneous lupus coinciding with a nonspecific pituitary lesion.

Of the first class I have nothing to say, except that it was not necessary to wait for an outbreak of cutaneous nodules to make the diagnosis of pituitary lupus.

Of the second class I will say that in all cases where it existed, the pituitary lupus plainly preceded by several months, sometimes by several years, the cutaneous lupus outbreak.

The third class is represented by one case of lupus of the cheek coinciding with a rhinitis, atrophic in type without ozena, and which I reported in the Annales of January, 1906.

Here my view seems at fault. There is, then, at least one case of facial lupus without any pituitary lesion. Now it is not proven that an affection so little understood as atrophic rhinitis is not a manifestation of tuberculosis. I gave to the French Laryngological Society of 1907[40] the clinical reasons favoring this hypothesis. Lombard, to whom I imparted my ideas as to the possible tuberculous nature of atrophic rhinitis, has obtained a general and local reaction by an injection of tuberculin in a patient having ozena. It is then not impossible that atrophic rhinitis may be a sclerotic tuberculous manifestation of the same sort as certain renal scleroses (Barnard and Salomon) and possibly hepatic scleroses. But, even if this opinion is not irrefutably demonstrated, the case of lupus of the cheek of which I have just spoken must be regarded as of pituitary origin.

We shall see further on that pituitary lupus spreads to the face by the lymphatic route. Now, it is perhaps not necessary for Koch's bacilli to produce a lesion at the point of inoculation in order to go thence to the lymph relays of the face. This explanation is not at all contrary to the general laws of pathology. It is positively known that Koch's bacillus penetrates the intestinal wall without making any lesion in it, and infects the mesenteric lymphatics, possibly also thus going from place to place in the lung. It is known that this same Koch's bacillus penetrates the tonsils in the same way, infects the tributary glands (Dieulafoy,[41] Babes[42]) and eventually the lung.

It follows from this that facial lupus would always be of pituitary origin, whether or not there be any lesion at the port of entry.

2. *Role of the lymphatic system in the appearance of facial lupus.*—It is curious to note that the places of election of facial lupus are just those points where there are ganglia, or lymphatic relays: Cheek bone (suborbital ganglion of Poincetreau); cheek (genial ganglion); horizontal branch of the inferior maxilla (pre-masseteric ganglion), these are for the lymph chain of the facial vessels. Ascending branch of the inferior maxilla (preauricular ganglion), submental region (submental ganglion).

A priori we would have a right to infer that the skin is infected through these ganglia or efferent vessels acting as intermediaries. Observation shows that just these ganglia are very frequently attacked in the course of pituitary lupus. I have long insisted upon this fact. We may conclude, then, that these ganglia play an essential role in the appearance of cutaneous lupus.

Arriving at the ganglion the bacillus may infect the skin and cause lupus in two ways:

(a) It may produce a suppurative adenitis, the fistula from which is the focus of the lupus (Cases VIII and XIII, lupus around a submaxillary adenitic fistula). The role of the lymphatic system is especially clear in the latter case. In fact, although the patient declared that the lupus nodules on the nasal lobule had been contemporaneous with the nasal obstruction and nasal crusts, the extent of the pituitary lesions proved that they came well before the cutaneous lesions. Now, considerably before the appearance of lupus in the lobule, the whole lymphatic chain of the facial vessels had been infected. Three years before my examination there was a suppurative right submaxillary adenitis treated surgically, and a suppurative submental adenitis. Ten months before, right suppurative premasseteric adenitis. Three or four days days before my examination, appearance of a tuberculous gummi in the skin, the size of a small pea, at the center of the cheek.

(b) The ganglion does not suppurate but the bacillus infects the afferent cutaneous lymphatics. I have been able in one case to find at the center of a lupus in the masseteric region (progressing to a cure, by the way) a small mobile ganglion, palpable proof of the mechanism of infection.

It is very evident that if instead of the ganglia described by Poincetreau, there exists only a lymphatic plexus, the latter would play the same role as a ganglion. If it were necessary to give other proofs of my ideas I could relate several other cases which I have had the good fortune to observe and which have the weight of laboratory tests. I have already related one of these cases in my note of January, 1906 (Case IV).

This was a young girl of 13, who, some time after a curettage of a lupus in the vestibule, noticed a lupus nodule appear on the left cheek bone, while at the same time, upon the horizontal branch of the inferior maxilla, same side, exactly at the spot occupied by the premasseteric ganglion, appeared a tuberculous subcutaneous ganglion, still mobile but rapidly adhering to the skin.

The second case, in every point comparable to the preceding, is that described as Case VI in this report Five days after a nasal curettage for a vegetating lupus there appeared redness of the cheek with characteristic lupus nodules, which were treated as such at the Hospital St. Louis, where I sent the patient. I have been able to see counterparts of these cases.

If a curettage of the mucosa can inoculate the skin of the face, inversely an amelioration of the nasal lesion can bring on a spontaneous disappearance of the skin lesions. This is what happened in the case above cited where the face lesions (lupus nodule and gumma) diminished gradually and disappeared without any special treatment other than that directed to the nasal mucosa.

Bresgen[30] has reported analogous cases of disappearance of face nodules after endonasal treatment. I have noted a complete concordance between the pituitary condition and that of lesions of the nasal integument, the latter growing better and even disappearing with pituitary improvement, reappearing when on account of insufficient care the pituitary lesions resumed their progress (Case III, *Annales,* January, 1906). Hence we can say that the state of the skin of the nose and face reflects the state of the nasal mucosa.

I have especially in view here lupus of the face, but it is very evident that lupus of the lobule is produced through the lymphatic openings in the skin.

I may add, in closing, that lupus may be inoculated into the skin without the medium of lymphatics. It sometimes follows the lacrymal canal and comes out upon the face through a fistula of the sac (Case II. *Annales,* September, 1906. See also cases of Hinsberg). Nevertheless this mode of propagation is more rare than the other.

It is not my intention to deny the possibility of direct infection of the skin. Clinically a previous skin lesion (eczema usually, but we must mistrust those eczemas which are sometimes tubercular manifestations) may favor contagion carried by kissing or rubbing.

Experimentally the bacilli can be made to enter the intact skin by friction (Babes,[42] Courmont and Lesieur[43]). But, for one thing, these experimental conditions are never realized clinically. For another, direct infection of the skin cannot explain the clinical peculiarities of facial lupus, especially its topography, which the lymphatic pathogenesis I advance explains so clearly.

B.—Lacrymal Complications.

Infection of the lacrymal passages in the course of pituitary lupus presents a quadruple character: frequent, early, caused by a simple or by a specific infection, tuberculous like the lesion which gave it origin.

It is frequent.—In twenty-two cases of pituitary lupus I have seen it eleven times. If I add two other cases of lupus which I have seen with lacrymal lesions and which were sent to me by Dr. Morax, we have a total of thirteen cases of lacrymal infection out of twenty-four pituitary lupus cases, or more than 50 per cent. I am not a little surprised to see this cause of lacrymation so infrequently mentioned in the French works upon lacrymal infections.

Raulin declares epiphora to be possible theoretically but he has not found it mentioned in the sixteen cases which he has collated.

Herzog notes several cases in his memoir.

Gelle[22] in his report to the French society noticed nasal tuberculosis but twice in 100 cases of tearing examined by him in Prof. Lapersonne's clinic. He cites statistics of thirty cases of tearing collated by Faravelli and Kruch without a single case of tuberculosis.

It is early.—In my eleven cases it appeared twice even before any nasal trouble had attracted the patient's attention; three times simultaneously with the nasal complaint, three times six months afterward, and once eighteen months afterward. In two cases the time of appearance was not indicated. Infection is therefore clearly contemporaneous with the beginning of the nasal lesion, and is indeed, as I say, a revelation sign. It is possible that the slightness of the lesion has caused it to go unperceived in a number of cases reported.

It may or may not be specific.—If it is a simple infection, inferior meatoscopy will show no lesion in the meatus proper. The lesion stops at the meatal floor. Or, it produces a synechia of the inferior turbinal border with the floor, favoring a relative stagnation of the tears and an ascending infection of the lacrymal passages.

On the contrary, if it is a tubercular infection of the lachrymal passages, meatoscopy often reveals characteristic lesions in the inferior meatus. Thus we may find a small lupus tumor in the meatus, seeming to come from the infundibulum (Cases I and II. *Annales,* September, 1906). Sometimes, too, the meatal surface of the inferior turbinate is involved, and fuses with the sinus wall, partly obstructing the inferior meatus. But the absence of a meatal lesion on meatoscopy does not absolutely prove that the lacrymal infection is of the simple sort. I have seen a case of lacrymation without a meatal lesion, which I first considered to be secondary and without significance, give rise later to fistula of the lacrymal sac and a lupus of the overlying skin.

It is therefore difficult to say in a case of tearing whether the lacrymal infection is simple or tubercular, and whether or not the patient is menaced by lupus of the sac integument.

C.—Sinus Complications.

Infection of the sinuses is possible in theory, and, as in the lachrymal passages, we can conceive of a simple infection of the frontal or maxillary sinus, manifesting itself by a catarrhal sinusitis, or a specific tubercular infection of the sinus mucosa.

I have never found signs of catarrh about the middle meatus. On the other hand, endoscopy would evidently not be informing as to the state of the sinus mucosa, for the infiltration of the mucosa would not be sufficient to intercept the light rays. I have used transillumination three times for the maxillary and frontal sinuses without getting indications of any lesion.

In fact, tuberculous sinusitis is not thus produced; it is always consecutive to a tuberculous osteitis adjacent.

However, Gleitsmann,[33] who has collected twenty scattering cases of sinusitis from the literature, reports (though without giving details) two cases of maxillary sinusitis resulting from pituitary lupus. Gradenigo at autopsy of a tuberculous lupus patient found the mucosa of the frontal and sphenoidal sinuses infiltrated and swollen (*Ann. des Maladies d'Oreille,* 1899, p. 93).

To sum up, the complication appears to be rare and will not hinder you.

D.—Aero-digestive Passages.

Localization in the cavum is an intermediate step by which lupus extends to the Eustachian tubes, the middle ear, the pharynx, velum and larynx. I will not expand upon lupus in these regions. That would be going beyond the scope of my subject.

I will only remark that these localizations are absolutely painless, having no symptoms or almost none. Hence it is necessary to examine these regions systematically in order to determine any extension thereto.

E.—Aural Complications.

The action of lupus upon the mucosa of the ear is exactly comparable to that upon the lacrymal passages. It may act in two ways:

(a) It occasions a simple infection which shows as a catarrh of the middle ear, exactly analogous to the lacrymation previously mentioned.

(b) Or it invades the tube and middle ear, produces lupus of the middle ear, dislocates the chain of ossicles, perforates the tympanic membrane and shows itself to the patient by a slight discharge from the canal.

The ear is frequently involved, for Felix[34] in twenty lupus cases seen at the dermato-syphilitic clinic of Bucharest found such lesions in every one; seventeen catarrhal and three suppurative.

The lesion must be sought out, for the impairment of hearing is often not enough to attract the patient's attention.

I have frequently found middle ear catarrh, and once (Case

XII) suppuration of the middle ear with total destruction of the drum except Shrapnell's membrane.

The prognosis is grave. On the one hand, treatment of the catarrh is often made difficult by the lesions in the nose and pharynx, partly because of the difficulty of introducing sounds and partly because of the constant reinfection of the tympanum. On the other hand, lupus of the tympanum may spread. Thus Gradenigo[35] has reported a case where the lupus had not only destroyed the ossicles and invaded the mastoid mucosa but had penetrated the labyrinth by breaking through the round window, occupying all the tympanic area. It had also invaded the oval window, and a little of the external semicircular canal. It was, as may be seen, a genuine lupus vorax of the ear. The type of lupus was very severe, having invaded the nasal fossae, the cavum with adhesion of the velum, the tongue, pharynx, larynx, and skin of the face, ear, body and limbs.

F.—Intercurrent Complications.

The most common is erysipelas. It occurs rather often in nasal lupus. Not uncommonly the patient may have in the long evolution of his case, several recurring attacks of erysipelas.

In a number of cases an amelioration of the lesions follows.

VIII.—DIAGNOSIS.

The diagnosis of lupus will be kept specially in view in this chapter.

Miliary tuberculosis has characters (form of ulceration, borders, vestibular seat, peripheral nodules, lively pain, rapid progress, advanced lung lesions) upon which I have dwelt sufficiently, and which in general are enough to make recognition easy. In what follows I suppose an isolated pituitary lupus, since a concomitant face lesion facilitates the diagnosis.

We may have to make a diagnosis in the presence of the following different conditions: beginning lupus, infiltration, perforation, tumor, vegetant lupus.

A.—Beginning Lupus.

The diagnosis is made by recognizing the elementary lesion. Remember that it is necessary to search systematically when a patient has one of the revelatory signs which I have pointed out, and to search in the places of election, upon the cartilaginous septum, the entire anterior portion of the subseptum and the floor of the meatus.

A very small error is possible; one must not take for a beginning lupus a swelling which is sometimes seen at the entrance of the inferior meatus, vaguely mammillated, due to a thickening of the maxilla. It will suffice to note that the mucosa which covers it is smooth and not roughened as in lupus.

B.—Lupus Infiltration.

This is very special; the mammillated infiltration is generally sufficiently characteristic. Several causes of error, though, are to be noted:

1. *Mammillated appearance of old nasal catarrhs.*—From time to time are seen old nasal catarrhs, with a kind of dermopapillary transformation, giving a mammillated aspect to the floor and turbinates. But, as in the preceding case, the mucosa preserves its smooth, polished appearance; further, it is not infiltrated and nowhere are there adhering crusts.

2. *Diffuse syphiloma of the nasal fossae.*—This is a variety of tertiary syphilis characterized by an often considerable infiltration of the septum and inferior turbinates, which really could not be confounded with lupus infiltration. The latter is mammillated, quite regularly mammillated; its progress is insidious and slow; it causes no annoyance to the patient except blowing out crusts. The nasal obstruction comes on with extreme slowness, often several years after the patient first noticed the crusts.

The infiltration of a diffuse syphiloma has opposite characters to those just described. It is smooth in its ensemble; there are only simple swellings, widely separated from each other, and a few ulcerations when breaking down begins. It comes on abruptly. In two or three weeks the septum and turbinate are quadrupled in volume, causing considerable nasal insufficiency. There is intense headache. Finally, the bones themselves are involved in the syphilitic process and are tender to pressure.

C.—Lupus Perforation.

Diagnosis may be required either of a lupus perforation in progress or of a healed perforation.

1. *Syphilitic perforation* is not easily confounded with lupus perforation. In lupus the perforation is seated only on the cartilaginous septum; in fact tuberculosis always respects the osseous structures. Of variable dimensions (a pea to a two-franc piece or larger), it is surrounded by a collarette of soft bleeding characteristic fungosities. Finally and above all it is never alone, the neighboring parts of the septum, the turbinates or vestibule, show the characteristic mamillation.

Syphilitic perforation is almost always in the bony septum. At that period rather often there are ulceration which upon being explored reveal denuded bone or sequestra. To this may be added the peculiar odor wholly unknown to lupus, the pain, headache, etc.

Exceptionally syphilis may attack the quadrangular cartilage. Considering the relatively rapid march of the lesion it is more likely to be confounded with lupus vorax. In both cases the perforation is rapidly effected. There may be a collarette of fungosities about the perforation and the lobule of the nose may be red and infiltrated. This is certainly a very embarrassing condition.

The diagnosis of lupus is to be made if there is a zone of mammillation about the perforation, if with the lymphangitis of the lobule there are characteristic nodules, if the gland territories of the nasal mucosa are the seat of adenitis.

But secondo-tertiary eruptions may simulate nodules of the lobule; there may be an adenitis in syphilis if there be a superinfection. So that often recourse must be had to specific treatment in order to settle the question.

When the perforation is cicatrized confusion with Hajek's ulcer is possible.

To tell the truth there is hardly any difficulty unless there be no other pituitary lupus lesions. Even in this event the borders of the ulcer would be a sufficient indication. In simple ulcer they are evenly thinned from the periphery to the center, and thin as paper immediately around the hole. In lupus they have a polymorphic aspect, thin in one place and thick in another, as though punched out.

D.—Lupus Tumor.

It is classical to say that the diagnosis of tubercular tumor is difficult, and that it may be confounded even with malignant tumors, especially sarcoma.

To my mind the difficulty is not so considerable. Distinction should be made according to whether the tumor is or is not accompanied by perforation of the cartilaginous septum.

1. *The tumor is accompanied by perforation of the cartilaginous septum.*—There should be no hesitation, it is lupus. Tumor and perforation were caused by the same thing. Only lupus can give this combination.

2. *The tumor is not accompanied by perforation of the septum.*—I repeat what I have just said; it is exceptional for the tumor to be the only manifestation without any other lupus symptom.

There is a hypertrophic variety of secondary pituitary lesion which has been described as condyloma of the nasal fossae and of which two cases have been reported by Lacoarret[31] and Labit.[32]

These growths do not seem to have a very clear symptomatology. In Lacoarret's case the tumor, which was implanted upon the septum, had dimensions of 1 cm. It was composed of three big mammillae and coincided with mucous plaques in the mouth and balano-preputial groove.

In Labit's case there were no concomitant syphilitic manifestations, but the tumor was hard and of horny consistence. Both tumors healed under specific treatment.

These two cases are rarities; they are the only ones I have found in the French literature. Hence there is no reason to give them an exaggerated clinical importance. Nevertheless they should make us prudent in diagnosis, and in case of doubt administer specific treatment.

All unite in saying that lupic tumor may be confounded with malignant tumor of the septum.

The question does not come up unless the patient is old (epithelioma) or when, with a middle-aged patient, specific treatment does not cause any change, the tumor not being accompanied by any other lupic lesion.

The latter condition is extremely rare. But when it exists it is very certain that, no more than other tumors of the nasal fossa, the lupic tumor has no objective character peculiar to itself. Then the microscope only, or, if necessary, an inoculation, can solve the question.

IX.—TREATMENT.

I am convinced, from the cases which I have treated, that general treatment is of the highest importance. It will not suffice alone to cure pituitary lupus but it puts the organism into a state where it can fight against the infected remnants which local treatment almost of necessity leaves behind, even after a most painstaking curettage. Cases are reported of curing lupus which had local treatment regarded as insufficient by the physician, but which several years afterward were seen in a state of cure under the sole influence of general treatment. Of course we cannot always count upon such marvelous results. But, I repeat, the first indications in treatment are superalimentation, fresh air and cod liver oil.

This said once for all, let us see what local methods we have at our disposal.

The methods employed can be classed as:

Physical.
Chemical.
Surgical.

Physical methods.—They comprise radiotherapy, hot air, phototherapy and cauterization.

Radium therapy.—This method is still in an embryonic state. It has been used by Botay[15] in one case. The method is too new to be passed upon. Of course it can only be used in the big cities.

Hot air.—Hollander ("Traitement de la tuberculose des muqueuses par les cauterizations a l'air chaud," Berlin Society of Medicine, May 16, 1906, in *Presse Medicale,* 1906, p 369), declares that he has gotten excellent results in this way. Combined with general hygienic and dietetic treatment, it will in the majority of cases, even the most advanced, almost always bring on a cure.

I have used this method only once, that was in Case VI. Treatments were given every eight days for a month, and hot air was applied to the lesions for five minutes. I then applied the canula directly to the lesions and made applications of six to eight minutes, hoping thus to get a cauterizing effect. After a month of this treatment the lesions were not influenced at all and I quit treatment. Other similar trials are of course necessary to judge of the method.

Galvano-cauterization.—This is an excellent method if too much is not expected of it. It is indicated in beginning lupus when the lesions are limited to the septum or the inferior meatus or the end of the turbinate. It is wholly insufficient for vegetant lupus.

Here is my method: Using a galvanic knife I make burns upon the infiltrated region, parallel to each other and covering the entire affected region. Over these cuts and at the same sitting I make another series of rays, parallel to each other, but perpendicular or oblique to the first cuts. This method, which is analogous to that employed by veterinarians for the treatment of joint tuberculosis, has given me better results than cautery points. The burn ought to be deep enough to go through the lesion.

Chemical methods.—Among these some are used directly, without any preliminary treatment of the lesions; others are used only after curettage. Among the first is cauterization with iodid of mercury; among the second is cauterization with lactic acid.

Iodid of mercury.—This proceeding has recently been recommended by Hollander (Berlin Society of Medicine, May 16, 1906).

He uses iodid of mercury in the nascent state. A quarter of an hour before the local treatment he has the patient take a dessert spoonful of 5 per cent iodid of potassium. Then he applies powdered calomel to the lesions, either on a tampon or by insufflation, or an oily emulsion. The iodine eliminated from the mucosa combines with the calomel and gives rise to iodid of mercury, whose action upon the lesions is most energetic. He has used this method not only in pituitary tuberculosis but also in buccal, vesical and laryngeal tuberculosis —the last with fine success. I have tried it in Cases I and VI, without any success. Maybe my technic was faulty, as I gave the iodid from one to three hours before insufflating the calomel.

Hirschberg used this method also with success in conjunctival tuberculosis.

Lactic acid.—This is only to be used after curettage.

It is very efficacious on the double condition of being employed in a very concentrated solution, 75 or 80 per cent, and of being used not by mopping but upon tampons left for 25 or 30 minutes. As long as I tried mopping I only had failures.

As soon as I began using the pack, results came with unexpected rapidity.

In the course of my research I have found that this method had already been employed by Hinsberg (*Arch. of Otology*).

SURGICAL TREATMENT.

This comprises ablation of the lesions with the bone adjacent and curettage of the degenerated mucosa.

Ablation of a portion of the septum has been done by Onodi in one case of primary tuberculosis (Onodi, *Arch. int. de Laryngologie,* November and December, 1906).

This operation is quite logical. Given the great frequency of septal perforation in lupus, is it not preferable to make a perforation right at the start, going beyond the disease limits and thus giving every possible chance of cure? Esthetic exigencies only permit doing this when the lesion is such that the subsequent perforation will not be visible externally.

Some authors (Tretrop, Delsaux, Belgian Laryng. Soc., 1906) have preceded the curettage by rhinotomy, stripping up the ala nasi (Delsaux) or by median incision (Tretrop).

In Delsaux's case the operation wound was infected, for lupic nodules appeared in the scar some months after the operation.

Leaving this possibility aside, I do not believe that a rhinotomy is really advantageous for a curettement of the nasal fossae, which can be done very satisfactorily through the natural way.

Is it necessary to chloroform the patient? I think that chloroform has two clear indications: considerable extent of the lesion and fearfulness of the patient. It is plainly indicated in cases of vegetant lupus involving both the middle and lower turbinates and the corresponding part of the septum. In this manner at a single sitting all the mucosal lesions, can be destroyed, leaving only remnants to be done away with subsequently by lactic acid dressings.

Likewise too sensitive patients are spared the torment of repeating curettages, which are always painful in spite of cocainization.

Summing up, the technic which I have adopted is:

Very limited lupus; cauterization.

Limited but vegetating lupus; curettage and lactic acid dressings.

Very extensive and vegetant lupus; curettage under chloroform.

The curettage should be painstaking to avoid recurrences. The granulations must be sought for in every place they may be, especially the edge of the vestibule, the angle between the edge and the ala nasi, and in the cul-de-sac of the vestibule, where they are easily hidden.

I apply the lactic acid dressings methodically by tamponing the nasal fossae. Between the inferior turbinate and the septum, whether these organs are involved simultaneously or separately, I introduce a wad of cotton dipped in a solution of lactic acid, 75 per cent. Two wads may be put in side by side if the space between the turbinate and septum is wide, so that the dressing will be in close contact with the lesion. A tampon may be placed in the inferior meatus if it is involved, and another in the middle meatus, above the others, if the middle turbinate is affected, always in such a manner that the dressing is in close contact with the turbinate, without compression however. The dressing is taken out after 15 to 30 minutes according to the endurance of the patient, and is repeated three times a week. The treatment is painful but bearable as a rule.

The results seem to me to have been remarkable, while they were nil with simple lactic acid moppings. The moment I began these dressings I had an improvement in Cases I, XI, XII, XIII and IX.

The mammillated infiltration disappeared very rapidly, leaving in its place a regular mucosa, smooth and of normal consistence.

The delicate point is to know exactly at what moment to stop the dressings.

It may be said that when there is doubt, the mucosa is still diseased. Every mammilla should be removed. The curette often gives valuable information by giving the soft sensation, characteristic and revelatory of lupus. In spite of everything the treatment is long and delicate. It requires much confidence and docility on the part of the patient, and much persuasion and tenacity on the part of the physician.

CASE I.

Pituitary Lupus. Lupus of Nose.

Mme. V., 29, came to Dr. Sebileau's clinic because of a constant discharge of crusts from both nares. She had no pain. But she feared that she had some serious trouble in the nasal fossae because of an extensive lupus of the nose now cured and dating ten years back. The entire external surface of the nose, almost to the root, was the seat of a thin bluish-white scar, traversed by fine capillary varicosities. The scar had been left by an extensive lupus which had produced one ulceration near the nasal bones proper, and two others about each naris which were deeply hollowed out.

This is her account of the appearance of the lupus:

After being exposed to severe cold (five hours in an open carriage, her body exposed to a biting December wind), she awoke in the morning with a swelling of the entire nose, extending to both cheeks. Some days afterward the swelling became localized to the nose, principally the nares, and became reddened. After a year ulcerations appeared. After being treated by scarification and cautery punctures without result, she went at the end of six years to Dr. Almagran, of Orleans, who cured her with sixteen x-ray treatments.

Now, five years before the nasal lupus, she noticed that the nasal secretion had become more abundant than normal. It also became dirty and slightly purulent, but had no odor.

When I first examined the patient, Sept. 1, 1902, I found: Small mammillae on the anterior portions of both turbinates, the size of large pin-heads, separated by minute ulcerated grooves. The mucosa about had lost its polish and was pale.

On the septum, anteriorly, was a mass of soft granulations the size of a pea, in the middle of which the probe penetrated the septum.

The patient's mother died of lung trouble at 24, the patient then being 4, two maternal aunts also died of tuberculosis. She had no lung lesion. Small submaxillary adenopathy, which had been much larger.

Removal of vegetations with curette and mopping with 20 per cent solution of lactic acid. Cauterization of the ends of both inferior turbinates.

In March, 1903, after several cauterizations, she seemed completely cured. However, in July, 1903, there was a small recurrence on the turbinal ends. Actual cautery.

In March, 1904, two cutaneous lupus nodules came upon the external aspect and free border of the right ala.

The pituitary lesions reappeared at the circumference of the septal perforation and the middle portion of the inferior turbinal border. For several months I watched the patient but one day she ceased to come.

I did not see her until June, 1905. The perforation was the size of a franc piece. It had fungosities, especially on the posterior border. Both inferior turbinates had lesions, but their ends were healed. The little betraying mamillae were about the middle portions.

For eight months actual cauterizations were made at periods varying from three weeks to two months.

December, 1905, for the first time, the left middle turbinate showed a mammillated aspect which left no doubt of its involvement. The mammillae were, however, flatter than those upon the inferior turbinate, and the grooves a little larger.

Not daring to cauterize the middle turbinate, I curetted (which showed lesions of characteristic softness) and mopped with 25 per cent lactic acid. Same operation upon the inferior turbinates and the perforation.

June 9, 1906, the periphery of the perforation and the turbinates appeared to be in good condition.

November 10, 1906. New fungosities on the upper and anterior edges of the perforation. The perforation was now the size of a franc piece. The posterior border was three times as thick as the anterior and had not gone beyond the limits of the cartilage. There was evidently a tubercular chondritis. The inferior turbinates seemed cured; they contracted a little under adrenalin. The cavum, tonsils and larynx were normal. Ears normal.

Seen again January, 1907. Mammillae have reappeared on the posterior margin of the septal perforation. Application of a cotton tampon wet with 75 per cent lactic acid for 20 minutes, repeated every other day.

February 6, 1907.—Perforation is cicatrized, anterior border thin, posterior thick. Turbinates smooth and seem completely cured.

CASE II.

Pituitary Lupus. Consecutive Nasal Lupus. Bilateral Epiphora.

Mme. P., 43, came to consult me at my Amsterdam St. Polyclinic, having been sent by an associate who had treated her for a nasal lupus involving the lower two-thirds of the nose. I first saw her Nov. 23, 1903.

The lupus had begun a year previously, in October, with two or three nodules on the lobule. For four or five years she had been annoyed by a nasal trouble which manifested itself by a discharge of crusts, sometimes bloody.

She had a bilateral epiphora which came at the same time as the nasal lupus.

Rhinoscopy.—Mammillated and grooved infiltration but not in as high relief as in the preceding case. The lesion has its maximum upon the antero-superior wall of the vestibule, on each side in the angle of the ala with the septum. In this region there are also a few soft fungosities which extend slightly to the adjacent part of the cartilaginous septum. The lesions are also present, on the head of the turbinates; on the right the latter is in contact with a deviation of the septum; on the left the lupus head of the turbinate has formed a synechia with the septum very near the floor; all the anterior part of lower turbinal border is adherent to the floor, so that the inferior meatus is transformed into an almost closed sac. Lungs healthy.

Epiphora on both sides but most pronounced on the left.

The lesions were treated exclusively by galvano-cauterization, repeated every eight days. December 23 there was marked improvement in the pituitary lesions, without any change in the lacrymation.

December 30.—The turbinates look much better; but the cartilaginous septum back of the vestibular angle is involved to the extent of a 20 centime piece.

January 10 and 20.—Mopping with lactic acid. Unfortunately she was obliged to return to her home, and I ceased to see her.

CASE III.

Pituitary Lupus Beginning in Tumor Form. Consecutive Nasal Lupus. Lacrymation.

Mme. M., 59, seen in Dr. Sebileau's clinic April 24, 1904 Eight years before, she noticed interference with nasal respiration accompanied by oozing at night. At this time a physician saw a tumor in the left nasal fossa which he removed. He afterward saw mammillations of the mucosa and thought it syphilis, then gave Gilbert's syrup.

At present she has the following cutaneous lesions: Ulceration of the left surface of the nasal lobule and adhesion of the left ala to the anterior part of the subseptum; lupus nodules scattered over the nose and dorsum, especially on the left, where they reach the nasal bones proper. It was only four years ago that the lobule of the nose began to "bloat," and redden. Three years ago the ulceration and lupus nodules appeared.

The epiphora of the left lacrymal canal appeared shortly after the nasal insufficiency.

Anterior Rhinoscopy.—Both sides of the nasal septum have a mammillated infiltration of a pale blue color. The lower part of the left side of the septum and the adjacent part of the floor are especially involved. The septum has a pale bluish infiltration in this region for its entire length, almost to the posterior portion, very prominent above the healthy mucosa and composed of a lot of minute granules separated by whitish grooves. Some of these granules are united in places making little vegetations which are flabby to the probe.

The middle portion of the infiltration is ulcerated, with scooped out borders.

The extreme anterior portion of the quadrangular cartilage has a very small perforation.

The right inferior turbinate is healthy as well as the middle turbinate, same side.

The left inferior turbinate is hard to see because of the adhesion of the ala to the lobule. It seems to have the mammillated infiltration

Lungs normal to auscultation. No tubercular antecedents.

Curettage of the infiltration and septal ulcer, which were very soft. The curettage brought out actual fungosities. The left inferior turbinate was also curetted and was equally soft. The curettage was followed by application of 20 per cent lactic acid.

Seen next, May 28, 1904. At St. Louis she had had the lobule of the nose curetted. The affection seems to have taken on a rapid course. The perforation is large and surrounded by a collarette of small friable fungosities. The right anterior portion of the septum has abundant granulations. The internal surface of the ala is much infiltrated.

But the lupus nodules of the nasal dorsum had disappeared. They reappeared later when the pituitary membrane got worse, to disappear finally and permanently with an improvement in the pituitary condition.

Curettage followed by application of 20 per cent lactic acid.

June 10 and June 23.—Another curettage of the septum and floor.

July 8.—The lupus has attacked the upper lip. The pituitary seems in good shape, except in the left vestibule. Curettage of the vestibule and application of lactic acid. Actual cautery to the lip. The patient was seen every two weeks for three months, when she was lost sight of.

February 25, 1905.—Left nasal fossa in good condition. Upper lip cured. But all the nasal lobule, or rather all the lower third of the nose, has a tubercular lymphangitis and the corresponding surface of the vestibule is vegetating. The posterior third of the left septal surface is pale as if macerated, and has a series of punctiform depressions. It is smooth and hard to the probe, and is not infiltrated. In spite of its evidently bacillary appearance and the coincidence of lupic lesions I gave specific treatment for fifteen days, without improvement.

Soon the lupus nodules appeared on the subseptum, and about the last of May it broke, weakening the lobule.

Superficial cauterizations with point on the subseptum and adjacent lip area during June and July.

September 13, 1905.—Subseptum much better. The lobule has hardly any lymphangitis. The lip is healed.

But, beside some fungosities on the lower border of the septum anteriorly, as well as the anterior border of the perforation, there was a mammillated infiltration of the left middle turbinate.

Curettage and application of lactic acid, 20 per cent.

February 14, 1906.—The lobule is weakened more and more, the subseptum being completely destroyed.

The patient was seen again March 2, 1907. The external appearance of the nose is still worse. The lobule is slightly adherent to the nasal spine. The left ala is inserted under the lobule to which it is also slightly attached. The skin of the lobule is clear of any lesion. But the vestibule is fungous. The inferior portion of the septum on the right is infiltrated back to the junction of the middle and posterior thirds. The inferior turbinate of this side is healthy. On the left there is a reappearance of lesions on the floor and middle turbinate. The left lower turbinate is atrophied.

New curettage as complete as possible. This time after the curettage a wad of cotton wet with 75 per cent lactic acid was put in and left for a quarter of an hour (the patient could not stand it any longer), and was repeated three times weekly.

CASE IV.

Primary Lupus of the Nasal Mucosa Without Coincident Skin Lesion.

Eloise B., 20, at the laryngologic clinic of Lariboisiere Hospital, March 20, 1904. Father and mother dead. Father died of bronchitis; drinker and high liver. A sister died at 20 and another at 17 of pulmonary trouble; two other sisters died at 6 months and 2 years of smallpox. She had always lived apart from her sisters.

Aside from an angina at the age of 10 she had never had any illness. She complained of difficult nasal respiration since her first year.

Rhinoscopy.—All the left vestibule is infiltrated and has soft vegetations. The septum is infiltrated with scattered vegetations the size of a lentil to a pea, finely lobulated. There is a loss of tissue extending well back. No denudation of bone perceptible to the probe. The inferior turbinate of the same side is involved, but, aside from the usual pallor of such lesions, it shows only small ulcerated grooves separating nonprojecting fine lobules. The floor has the same lesions as the turbinate. No perforation of septum. Right nasal fossa clear. No odor, no headache, no pain upon pressure over the nasal bones, no spontaneous pain. No stigma of hereditary syphilis.

Four left submaxillary ganglia the size of a pea to a nut. Similar right submaxillary adenitis.

Lungs normal.

Inoculation of a guinea pig with a bit of the lesion—positive.

Lesions curetted, followed by mopping with 50 per cent lactic acid, then a series of galvanic cauterizations.

April 20, considerable improvement. Unfortunately the patient quit coming.

CASE V.

Pituitary Lupus; Consecutive Nasal Lupus.

Maria E, 16, came to the laryngologic clinic at the Lariboisiere, where she had been sent to me by Dr. Balzer, April 1, 1904. She had a nasal lupus which had been treated at St. Louis and which had originated six months before.

But pituitary lesions considerably antedated the cutaneous lesions, since for at least three or four years, she said that she began to have trouble in the right naris, blowing out crusts or picking them out with her fingers. Two years ago the same symptoms appeared in the left naris.

Anterior Rhinoscopy.—Pituitary lupus, characterized by mammiliated infiltration, not much elevated above the healthy mucosa, of pale red color and lacking luster. The infiltration is especially vestibular, but it also exists on the anterior fourth of the septum and inferior turbinates, with predominance on the right. The subseptum near the lobule is likewise involved.

Numerous submaxillary ganglia. The apex of the left lung shows perhaps a slightly harsh vesicular murmur.

I omit the past history which has no essential importance here.

April 8, 15 and 22.—I made a curettage of the lesions followed by the application. At the last visit a nest of nodules in the vestibule was discovered below the tip of the lobule, with a tendency to ulceration of the septum.

May 11.—Septum cicatrized; a few suspected nodules on the right inferior turbinate and the left floor near entrance. Curettage and mopping with lactic acid.

June 14.—Another curettage of the right inferior turbinate.

June 16.—Septum completely cicatrized. The floor on both sides had suspected points which were curetted then mopped with pure lactic acid. The general condition is also better. The patient is plainly gaining weight.

The submaxlllary adenitis has almost disappeared. The patient, who does not live in Paris, was obliged to return home.

CASE VI.

Primary Vegetating Lupus. Secondary Lupus of the Cheek Bone Following Curettage of the Mucosa. Left Lacrymation.

Mme. B., 42, came to the clinic at Lariboisiere November 10, 1905, for pronounced nasal obstruction.

Since the age of 10 she has been subject to coryzas lasting two or three months accompanied by a discharge of bloody crusts and disappearing in good weather. Two years ago she had been bothered by cacosmia which led her to consult a specialist, who cauterized with silver nitrate followed by application of tincture of iodin and the cacosmia disappeared in two months. He also curetted one of the nasal fossae.

A year ago the nasal fossa was curetted at Saint Antoine.

Four children living, well. Two children dead, one at 18 months of meningitis, the other of capillary bronchitis following measles. No miscarriages; no stigmata of syphilis. The lungs are sound. She has an epiphora of the left eye as a result of an abscessed lacrymal sac sixteen years ago. Did the lupus date back this far? Right and left submaxillary adenitis, voluminous in size, especially on the right, which she claimed to have had since infancy.

Rhinoscopy.—On the right the inferior turbinate is transformed into a pale vegetating mass, in contact with the septum The mass gives the impression of something finely stippled. The vegetations are composed of a multitude of fine granulations, and are separated by depressions or even losses of tissue. The ensemble recalls the raised school geography charts. The lesion seems to go very far back, but it is impossible to say definitely because the turbinate is in contact with the deviated septum. Same lesions on floor and internal surfaces of the ala nasi on this side. On the left, same lesions.

The cartilaginous septum has a mammillated and grooved infiltration, pale, bluish, and soft to the probe. Perforation far anterior. The middle turbinates are hard to see. After cocainization curettage of the right nasal fossa, then application of 30 per cent lactic acid.

November 16.—Curettage of left nasal fossa. When she came again on the 22nd, four days after the curettage, she had a lymphangitis over the left cheek bone with two lupus nodules. This lesion has since progressed like a lupus of the cheek.

There is no doubt in my mind that my curettage was the cause of the cheek lupus. So for a time afterward I used cauterization exclusively. But by this method the treatment took too long and I was obliged to go back to curettage, in spite of its dangers.

April 20.—Careful curettage of the right inferior turbinate and septum of same side, followed by application of 30 per cent lactic acid.

May 10.—Curettage of right inferior turbinate and septum. Beginning May 25 I cauterized the nasal fossae alternately every week until the end of June.

I saw the patient again July 20. Much improvement; the right inferior turbinate is almost healed. The left inferior turbinate is less swollen. But coming off from the left middle turbinate is seen a pale granular intumescence, soft to the probe. Both nasal fossae are completely permeable, a result which pleases the patient because they were entirely obstructed before treatment. Some fungosities around the septal perforation. Cautery stripes to the turbinates and points to the septal fungosities.

Seen again November 16, 1906. The anterior portions of both inferior turbinates are still diseased, as also the periphery of the perforation. Curettage and mopping with 50 per cent lactic acid.

January 9, 1907.—The posterior part of the left middle turbinate is enormously hypertrophied. There is a tumor hanging from it like a big raspberry, of a pale blue tint, lobulated and descending almost to the floor. The lupus of the cheek continues its course and is being treated at St. Louis.

Ablation of the tumor. Beginning at this time 75 per cent lactic acid packs were commenced. These were allowed to remain 25 minutes, and were repeated every eight days. Unfortunately after two treatments the patient quit coming to the clinic.

Result.—Patient much improved but not cured.

CASE VII.

Pituitary Lupus. Consecutive Lupus Over Left Cheek Bone and of the Cheek near the Horizontal Branch of the Left Inferior Maxilla. Bilateral Lacrymation.

Marie P., 26, came to the clinic November 4, 1905, sent to me by Dr. Balzer, in whose clinic she had been treated two years for lupus over the left cheek bone and left horizontal branch of the inferior maxilla. There were also healed lupus lesions of the lobule. The beginning of the affection was six years before. At that time she blew out crusts and had "growths of flesh" in the vestibule. Six months afterwards came the tearing of the right eye and only then the nasal lupus. Three children, well. Father and mother well. Lungs sound.

Rhinoscopy.—At present the left ala is mostly eaten out but has no lupus nodules. Left, mammillated, pale infiltration of the inferior turbinate. A few millimeters from the anterior end it has a synechia with the floor. (This is the side on which the epiphora is most pronounced.) The whole turbinate is infiltrated and has losses of substance in places. Right, the inferior turbinate has the same mammillated lesion especially along the inferior border, which is covered with purulent crusts. On both sides the inferior turbinates are greatly atrophied and the mammillae and grooves have the rifled appearance before mentioned. On both sides the middle turbinates are hypertrophied and have a very clear mammillated and grooved appearance. There is a small hypertrophied ganglion at the end of the greater cornu of the hyoid bone, left side. The treatment consisted of curettage and application of lactic acid, alternating with galvano-cauterization.

She quit February 22, 1906, very much improved, but not cured.

CASE VIII.

Pituitary Lupus. Consecutive Nasal Lupus. Suppurative Cervical Adenitis. Suppurative Submental Adenitis with Lupus Transformation of the Adjacent Skin. Lymphangitis of Nose. Scrofulous Habitus.

Mariette R., 19, came to the clinic at Lariboisiere January 6, 1906. Mother died of hepatic cancer with ascites. Father living and well. She has cervical cicatrices, right and left, from former suppurative adenopathies, especially behind the suprahyoid region. Occupying the entire submental region is a cutaneous lupus, developed upon a suppurative adenitis which opened spontaneously.

Three years ago she had some embarrassment of nasal respiration. She was seen at this time at Lariboisiere where a diagnosis of septal polypus was made; it was removed and the point of implantation cauterized. It was evidently a lupus tumor, as proved by the course of the case. Six months afterward there appeared two lupus nodules on the lobule, diagnosticated and treated by Dr. Jonselme, who cauterized them and brought about a cure. It was about this time that the submental adenitis developed lupus. At no time did she have nasal pain.

At present the nasal lobule is somewhat puffy, and slightly red at the tip. There is a small nodule on the vestibular border of the lobule on the left side.

Rhinoscopy.—Characteristic mammillated infiltration, nonvegetant. Right: The lesion occupies fully the anterior third of the inferior turbinate, the corresponding part of the septum, especially its vestibular portion where there is a tendency to perforate, and upon the point of the septum near the lobule. Left: Lesions less advanced; they hardly comprise the anterior third of the turbinate. The septum is involved only at its anterior portion by lesions from the lobule. On both sides the floor adjacent to the turbinates is diseased. No premasseteric adenopathy. Cavum, pharynx and larynx normal. Lungs sound. General condition very good; florid appearance. Not daring to curette for fear of making inoculations of skin lupus, I used the cautery every week in each nasal fossa, making streaks parallel to each other and crossed by other parallel streaks perpendicular to the first. February 16, 1906, the pituitary lesions are improving. But there is a lupus nodule on the posterior pillar of the velum near the upper pole of the tonsil, which I cauterized. Patient lost to sight.

CASE IX.

Pituitary Lupus. Lupus of Nose. Lupus of Upper Lip. Right and Left Cheek Bones. Right Epiphora.

Gabrielle B., 38, came to Lariboisiere February 24, 1906. She had a characteristic lupus of the nose. This had begun two years ago with two or three nodules on the upper lip at the entrance of the right naris, which spread to the right ala, then the lobule and finally almost the entire nose. Ten years ago there was epiphora of the right eye and at the same time some interference with respiration on this side. I do not hesitate to regard this as the origin of the present pituitary lesions.

Rhinoscopy.—Pronounced mammillated and grooved infiltration of the right cartilaginous septum as well as the head of the right inferior turbinate and of the vestibule at the junction of septum and left ala. In the septal infiltration is a loss of tissue the size of a fifty centime piece, with irregular borders and granular bottom. No denuded bone. No lesion in left fossa. Two weeks ago six nodules appeared over the right cheek bone. Cavum, larynx and lungs normal.

CASE X.

Pituitary Lupus. Lupus of Nose.

M., boy of 14, seen at my clinic at the Amsterdam St. Polyclinic, October 19, 1906. He has a lupus of the nose occupying much of the dorsum. Eight years ago he had lupus over the right cheek bone, which was treated unsuccessfully by radiotherapy and then excised by Dr. Nelaton, who made skin grafts from the thigh. At that time the patient complained of nasal obstruction and had pituitary lesions, for Dr. Nelaton at the time of the lupus excision curetted the pituitary.

Rhinoscopy.—Right fossa: The head and anterior third of the right inferior turbinate is transformed into a pale fungous tissue, formed of small mammillae separated from each other by minute, ulcerous grooves, bleeding slightly on being touched with the probe and not contracting under cocain. These lesions extend diminishingly to the posterior third of the turbinate. Same lesion on the middle portion of the cartilaginous septum. These lesions are difficult to make out because of a marked atresia in the right naris due to old vestibular lesions now cured, and perhaps to former curettages. Left: The inferior turbinate has a synechia anteriorly with a spur on the septum. There is a slight mammillation of the head. The synechia bears witness to old lesions. No bone lesion.

The velum, pillars, tonsils and posterior wall of the buccal pharynx have lupus with marked infiltration. It is impossible to see the cavum. The larynx is normal. Slight submaxillary and carotid adenitis. The patient does not cough. The lungs show no physical signs.

After cocainization I at once curetted the right inferior turbinate and septum. The lesions had a characteristic softness especially the anterior portion of the inferior turbinate. Application of 30 per cent lactic acid. Same operation on the left. The lesions are much less extensive, both as to surface and depth. Galvano-cautery points every week to the velum, pharynx and tonsils. The lesions on the turbinate and septum were treated alternately with cauterizations and curettages followed by mopping with 50 per cent lactic acid.

Beginning December 21, 1906, after a curettage of both inferior turbinates and the septum I used dressings once a week of cotton wads soaked with lactic acid, 75 per cent, which were left in twenty-five or thirty minutes. I would have liked to use dressings much oftener, but the patient would only submit to it once a week.

December 28.—The change is striking. The mucosa of the left turbinate seems to be healed. That of the right turbinate and septum, is for the most part, smooth and even except in the extreme anterior portion. Another 75 per cent lactic acid dressing, left in twenty-five minutes.

For the first time the laryngo-pharynx, which had been immune, now has six large mammillated infiltrations on the left. The left aryteno-epiglottic fold also has three lupus nodules, distinct and red, which had not previously existed. Cautery.

January 4, 1907.—Renewed application of lactic acid to the right inferior turbinate and septum for twenty-five minutes. The left turbinate no longer has any lesion. The lesions on the dorsum, which were treated at St. Louis with Finsen rays and cautery alternately, are much better.

March 12.—There remained only a few minute mammillae on the head of the right inferior turbinate.

The velum, pharynx and larynx are much improved.

CASE XI.

Pituitary Lupus Beginning on the Subseptum. Destruction of a Large Part of the Cartilaginous Septum and Both Ala. "Death's Head" Aspect. Lupus of Both Cheeks. Bilateral Epiphora.

Marie R., 46. The lupus of the nose began on the extreme anterior portion of the lobule. But, a year before the lobule lupus began, she had bloody crusts in the vestibule near the sub-septum. After the cutaneous lupus on the lobule came the lupus of the cheeks, first the right then the left. At present the ala and lobule have completely disappeared, the cartilaginous septum shows vertically between the mutilated external nasal walls—the "death's head." The entrance to the nasal fossae is narrowed by scar tissue. No pituitary lesions now. Cavum, larynx and lungs normal.

CASE XII.

Pituitary Lupus. Consecutive Lupus of Nose. Lesions of Middle Ear.

Louise B., 21, seen December 16, 1906, for nasal trouble characterized by obstruction and discharge of bloody crusts. The lupus of the nose appeared at the age of 16, in the form of five nodules on the lobule and extended to the right ala. After having had a considerable nasal discharge since the age of 10, she states that between 13 and 15, before the skin lupus, adherent sanguinolent crusts appeared upon the margin of the naris. At present the lupus of the nose is cicatrized. The right ala is partly destroyed.

Rhinoscopy.—The left inferior turbinate is somewhat atrophied, mammillated and ragged throughout. The middle turbinate also has ulcerated grooves. The septum, over almost its entire extent, has little pale infiltrated masses, composed of very small white points separated by ulcerous grooves. Smaller mammillae disseminated all over the septum. At the middle of the septum one of these masses comes in contact with a mammilla on the inferior turbinate.

Right: The head of the inferior turbinate seems involved, but the lesions are difficult to see because of narinal atresia and a deviation of the septum. No bone lesion. Both ears suppurating. The left was attacked with acute otitis media, following, it seems, an effort to expel crusts by blowing the nose. A mastoiditis followed for which I operated five years ago.

Both suppurating ears show identical lesions. Destruction of all the membrana except Shrapnell's. The mucosa is red and infiltrated. Slight amount of secretion.

Cavum.—Some mammillations on the upper surface of the velum, with crusts in the cavum. The facial sinuses are quite clear. Pharynx, larynx and lungs normal. After curettage under cocain I made tri-weekly application of 75 per cent lactic acid; leaving it in half an hour. February 11, 1907, the lesions seemed cured. Turbinates and septum are smooth and free from crusts.

CASE XIII.

Pituitary Lupus, Rapidly Progressing. Consecutive Nasal and Submental Lupus.

Alphonsine L., 19, was seen by me at Lariboisiere January 3, 1907. She has a nasal lupus occupying the lobule, ala and lower two-thirds of the dorsum. It is a lupus excedens with very prominent confluent nodules. Without the speculum it can be seen that all the vestibular part of the septum has been destroyed. The lobule is attached to the inferior border of the pyriform orifice only by a thin bit of cutaneous subseptum, which is also infiltrated with lupus and ready to slough off.

The beginning dates back four years. At this time there was a small swelling in the nasal fossa near the orifice which tempted the patient often to rub it. Eighteen months ago the superficial parts were attacked, and at the same time there appeared a suppurative submental adenitis which is now of a lupus character. No submaxillary, genial or premasseteric adenitis.

Rhinoscopy.—Extensive destruction of the septum comprising all the cartilaginous part, and making a perforation the size of a two-franc piece. In front it is bounded by the cutaneous subseptum, itself lupic, and below by the floor of the nasal fossae. The inferior turbinates are considerably atrophied, to a degree suggesting atrophic rhinitis. But the turbinates which are pale, are covered for the most part with pale red mammillae the size of two pin-heads, and separated from each other by grooves, some ulcerated, some cicatricial. Anteriorly in the floor of the left nasal fossa is an eminence as large as a nut, of a pale, bluish red and very delicately mammillated, of a framboesic aspect, soft, and sessile upon the floor. In the median submental region are two erythematous plaques with lupus nodules which succeeded a suppurative adenitis, as stated. Summing up, this is a lupus with relatively rapid course without being exactly lupus vorax. Pharynx and larynx normal. Lungs healthy.

Ear.—Diminution of hearing without suppuration or tympanic alteration. Maxillary and frontal sinuses quite translucent.

Hereditary antecedents.—None.

Personal history.—At 16 had pleurisy; at 17 eruption of furuncles on legs, in ear, on chin.

January 15.—After mopping with cocain I curetted out the large mammillated mass. Part of this mass was certainly fibrous, for, besides the soft lupus lesions there was hard tissue which resisted the curette. Dressing with cotton wads dipped in 75 per cent lactic acid left in half an hour.

January 25.—Curettage of the inferior turbinates followed by the same dressing. Three times a week the dressing was renewed and left in thirty minutes. The result was excellent and quick.

February 15, 1907.—Only a month after beginning treatment, the mucosa of the floor and both turbinates was smooth and appeared to be cured.

March 9.—The fragment of subseptum has become destroyed and the lobule is sunken.

March 15, 1907.—Cure is maintained save on the posterior border of the perforation where a few granulations have reappeared. Renewed application of lactic acid.

CASE XIV.

Infected Pituitary Lupus. Nasal Lupus.

Madeleine M., 14, came to the clinic January 10, 1907. She has lupus of the lobule, of the subseptum which is destroyed, and of the alae which are also mostly destroyed. The cutaneous lupus appeared only six months ago but the lesion had commenced a year ago in the "interior of the nose" where she saw "little balls."

Rhinoscopy.—On the cartilaginous septum, the inferior turbinate anteriorly and the floor, especially the left side, there is a mammillated infiltration with a vegetating tendency.

These pituitary lesions as well as the nasal ones are manifestly infected. On the nose the nodules are turgescent and the surrouding lymphangitis is considerable and quite red. The nose is very tender to touch. Same inflammation of the pituitary lesions which are red and tender to touch. There is an abundant secretion of mucus giving the lesions a varnished appearance.

For ten days I contented myself with lavages of sterile water and applying a dermatol ointment in the nasal fossae. After the lesions had about lost their inflammatory character, I curetted them under cocain, following with dressings of 75 per cent lactic acid, left in half an hour and repeated three times weekly.

March 9, 1907.—The condition of the right nasal fossa is almost unrecognizable; the septum is smooth; the inferior turbinate and floor are likewise smooth and healed. On the left there are still a few minute lesions.

The lungs, which were auscultated several times, seem sound. Transillumination of the face cavities never showed anything abnormal.

CASE XV.

Pituitary Lupus. Nasal Lupus. Tuberculous Gummi of Cheek. Suppurative Submaxillary Adenitis Causing Lupus of the Region. Submental Adenitis. Suppurative Premasseteric Adenitis.

Louise J., 30, seen February 12, 1907. She had lupus of the nasal lobule. There was considerable reddened infiltration upon which, at the upper portion, were situated seven or eight very prominent nodules. She declares that the lupus appeared only three months previously and at the same time the nasal obstruction and crusts in the right nasal fossa. The nasal lupus began with a little nodule above the right ala, near the right border of the pyriform orifice. From thence the lupus rapidly extended to the lobule. All the right side of the nose is infiltrated but without change of color. Below, the lesion has extended to the subseptum and that part of the upper lip in contact with the right naris. Although the patient (who is not very intelligent) states a simultaneous appearance of the cutaneous and pituitary lupus. I do not believe her for the following reasons: In the first place, the pituitary lesions are far too extensive to have lasted only three months. Further, well before the appearance of the nasal lupus, all the lymphatic chain accompanying the facial was infected, proving that lesions already existed about the anterior end of the nasal fossa. Thus it was that three years previously she had a right submaxillary adenopathy, treated surgically, and a submental adenitis which suppurated. Lastly, in April, 1906, there appeared in the region of the right premasseteric ganglion a reddened nodular swelling which suppurated, leaving in its place a scar the size of two half-franc pieces.

At present the old right submaxillary cicatrix is lupic. On a line uniting the naso-genial groove with the maxillary insertion of the masseter and slightly nearer the masseter than the groove, is an inflammatory nodule encased in the skin, the size of a small pea, jutting out and surrounded by a small inflammatory zone. This nodule, situated upon the lymphatic territory of the right facial, had appeared only three or four days before.

Rhinoscopy.—The maximum of lesions is on the right cartilaginous septum, which anteriorly has a mammillated thickening the size of a franc piece, jutting out several millimeters beyond the healthy mucosa. The mammillations, which are pin-head sized, separated by filiform grooves, group in places to form small granular bunches, separated by depressions from other bunches. The whole greatly resembles a relief geography chart, to which I have already made comparison. The lesion is a pale rose and comes in contact with an analogous and very pronounced lesion on the right inferior turbinate. For this reason it is impossible to see the middle turbinate. On the left there is a slight lesion of the inferior turbinate. No bone lesion. No lesion in cavum or larynx. The frontal and maxillary sinuses are very translucent. The patient coughs. She is subject to bronchitis. Auscultation seems to indicate a slight induration in the right apex.

February 20, 1907.—Curettage of the right surface of cartilaginous septum after cocainization. Unfortunately the patient is very timid and my cauterization was not at all thorough. Dressing of 75 per cent lactic acid, twenty-five minutes.

March 1, 1907.—Another attempt at curettage of the right inferior turbinate after cocainization. But I could not do it well because of the patient's intractableness.

March 9.—The middle turbinate was found to be not intact. Another fruitless attempt at curettage. Curettage under chloroform was decided upon.

CASE XVI.

Primary Pituitary Lupus. Epiphora.

Mme. X., 52, came to the Lariboisiere clinic February 5, 1907, for nasal trouble, chiefly on the left side, and characterized by a rather abundant production of crusts. She complained of lachrymation which had lasted seven or eight years. The beginning of the nose trouble dated back five or six years. Three years ago she had had a curettage of the right nasal fossa at Lariboisiere.

At present there are bilateral lesions of the cartilaginous septum. On the left is a round ulceration a little larger than a half-franc piece, flat, resting upon an infiltration which sticks out several millimeters from the healthy mucosa. The bottom of the ulceration is pale and dull. The infiltration is mammillated. Anteriorly upon the infiltration are small pimples separated by deep ulcerous grooves which may presage a perforation. On the right the lesions are of the hypertrophic type. There is a granular infiltration the size of a nut which almost touches the inferior turbinate. No lesion of pharynx, tonsils or velum. Lungs sound. Sinus quite translucent. No cutaneous lupus. Upon palpation there is felt a small, hard, premasseteric ganglion, the size of a small pea, mobile under the finger.

Inoculation of a bit of pituitary tissue in a guinea pig. A chancre developed at the point of inoculation, with caseous inguinal adenitis.

REFEFENCES.

1. Cartaz. France Medicale, 1887.
2. Boutard. La tuberculose nasale, Thèse Paris, 1889.
3. Plicque. La tuberculose nasale, Ann. mal. oreille, etc., 1890.
4. Olympitis. La tuberculose nasale, Thèse Paris, 1890.
5. Koschier. La tuberculose nasale, Wien. klin. Woch., 1895.
6. Pistre. Les pseudo-polypes tuberculeux des fosses nasales, Thèse Bordeaux, 1903.
7. Raulin. Lupus primitif des fosses nasales, Thèse Paris, 1887.
8. Hinsberg. Affections oculaires en relation avec la tuberculose de la muqueuse nasale, Arch. of Otology, 1901.

9. Massei. Rev. hebd. laryng., March 11, 1905, No. 10.
10. Escat. Legitimite de la distinction clinique du lupus, etc., Ann. mal. oreille, October, 1905.
11. Demme. Zur diag. Bedeutung die Tuberkelbacillen, etc., Berlin klin. Woch., 1883, xx, No. 15.
12. Volkmann. Ueber den Character der fungosen Gelenkentzündungen, Samml. klin. Vortraege, Leipzig, 1875-1880; Chirurgia, Nos. 168, 169.
13. Caboche. A propos de la tuberculose nasale, Ann. mal. oreille, January, 1906.
14. Marschik. Wien. klin. Koch., 1905, xviii, 22.
15. Heinde. Wien. klin. Woch., 1905, xviii, 22.
16. Caboche. Tuberculose nasale et meat inferieur, Ann. mal. oreille, September, 1906.
17. Milian. La sclerodermie tuberculeuse, Soc. med. hop., March, 1907.
18. Francis Steward. Tuberculosis of the Nasal Mucosa, London Guy's Hospital Rep., 1897.
19. Willigh. Prager Vierteljahrschrift, p. 4.
20. Weichselbaum. Centralblatt f. klin. Chir., 1882, p. 26.
21. Melzi. Arch. internat. laryngol. ital., etc., July and August 1904.
22. Gelle. Role des lesions nasales dans la pathogenie du larmoiement, Soc. Francaise Laryngol., 1903.
23. Poulard. Adenopathies dans les affections oculaires, Thèse Paris, 1903.
24. Menier. Manuel des maladies des fosses nasales, Paris.
25. Chiari. Maladies du nez, Breyre translation, 1905.
26. Molinie. Med. medicale, 1893.
27. Audry. Journal des malad. cut. et. syph., 1896, p. 729.
28. Mineau et Freche. Origine nasale du lupus de la face, Annales Dermatologie, 1897.
29. Sticker. Versamml. Deutsch. Naturforschen med. Aertze, Düsseldorf, September, 1908.
30. Bresgen. Deu. med. Woch., 1887, No. 30.
31. Lacoarret. Rev. hebd. de laryngol., 1892, p. 405.
32. Labit. Rev. hebd. de laryngol., 1895, p. 509.
33. Gleitsmann. Tuberculose des sinus accessoires du nez, Rev. hebd. de laryngol., June, 1907.
34. Felix. Participation de l'oreille moyenne dans le lupus des fosses nasales, Ann. mal. oreille, March, 1903.
35. Gradenigo. Gazetta degli Ospitali, 1888, in Annales mal. oreille, 1889, p. 93.
36. Pasch. Contribution a la clinique de la tuberculose nasale, Arch. f. laryngol., Vol. xvii, No. 3. in Ann. mal. oreille. November, 1906.
37. Mygind. Le lupus de la cavite nasale, Arch. f. Laryngologie, in Ann. mal. oreille, November, 1906, p. 493.
38. Gelle. Adenopathies dans les affections des fosses nasales Report to the Societe Francaise de Laryngologie, 1905.
39. Garel et Collet. Tumeurs de la cloison nasale, Ann. mal. oreille, 1893, p. 249.
40. Caboche. L'ozene forme larvee de la tuberculose, Sos. Francaise de Laryng., May, 1907.
41. Dieulafoy. Academie de Medecine, 1895.
42. Babes. Penetration des bacilles tuberculeux par la peau, Presse Medicale, June 15, 1907.

43. Courmont et Lesieur. Penetration des bacilles tuberculeux par la peau, Presse Medicale, 1907, No. 51.

44. Marc Andre. Lymphaliques des fosses nasales, Thèse Paris, 1905.

45. Botey. Ann. mal. oreille, August, 1906, Case VII.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINOLOGIC AND LARYNGOLOGIC LITERATURE.

I.—EAR.

On Vertigo and Disturbance of Equilibrium in Non-Suppurative Diseases of the Internal Ear.

WITTMAACK, Greifswald (*Archives of Otology*, Vol. xxxvi, No. 5). The functions of the labyrinth are for the cochlea the perception of sound, and for the semicircular canal apparatus the preservation of the bodily equilibrium. To diagnosticate disease of the labyrinth, there should be a functional disturbance in the sound-perceiving as well as in the apparatus for preserving the equilibrium. The anomaly of pronounced disturbance of hearing with the absence of all disturbance of equilibrium in many cases of nerve deafness is explained by a case of the author's where in complete absence of symptoms on the part of the semicircular apparatus, in a few weeks the patient became completely deaf and at the acme of the disease a pathologic examination showed a degenerative neuritis localized in the cochlear branch of the auditory nerve. These isolated affections of the cochlea nerve are relatively frequent while isolated disease of the vestibular nerve is very unusual, in fact there is no reported case of disturbance of equilibrium related to the semicircular canal system without simultaneous disturbance of hearing.

A symptom always suggestive of disease of the sound-perceiving apparatus, especially of the auditory nerve, is tinnitus which must fulfill these conditions; the tone must be high, continuous and very loud.

Vertigo as an aid to the diagnosis of disease of the semicircular canal apparatus can be regarded only when in the form of typical rotatory vertigo.

The reason why the auditory nerve is peculiarly affected in general diseases is explained on anatomical grounds. The cochlear branch with its associated cochlear ganglion is imbedded in an unyielding bony capsule partly surrounded by broad lymphatic spaces. The blood vessels which nourish this structure are end arteries, hence the vulnerability of the cochlear nerve to toxic substances or to any interference with the circulation. *Campbell.*

On the Surgical Exposure of the Jugular Bulb.

Voss, Berlin (*Archives of Otology,* Vol. xxxvi, No. 5). The author has observed in many specimens that the jugular fossa is not found at the floor of the tympanic cavity but near its median wall and that the bulb is not infrequently situated far backward near the mastoid. In such specimens the floor and the median wall of the tympanum as high as the promontory were made up of bone which separated the jugular fossa and the carotid canal.

After completing the mastoid operation, he exposes the sinus and removes the overlying bone downward to the lowest part of the vein. He uses biting forceps and narrow concave gouges so as to avoid injuring the facial nerve. The bulb is next exposed, and to escape the horizontal semicircular canal the bone is not removed higher than 0.5 cm.

Where there is doubt as to the bulbar contents, it can be determined by aspiration; when diseased the wall is incised and the thrombotic masses removed by forceps and curettes aided by irrigation. By removal of the lateral wall of the sinus and the posterior wall of the bulb the vessel is converted into a gutter easily accessible to treatment. *Campbell.*

On Chronic Progressive Labyrinthine Deafness.

Manasse, Strassburg (*Archives of Otology,* Vol. xxxvi, No. 5). In an anatomic investigation of thirty-one labyrinths the author found:

1. Corti's organ, including the membrana tectoria was atrophic to a stage of complete aplasia at the cochlear duct and its immediate surroundings; dilatation or constriction of the duct by distension or collapse of Reissner's membrane, hydropic degeneration of the spiral ligament, connective-tissue new formation at the lower extremity of the stria vascularis, connective-tissue and bone formation in the perilymphatic spaces, occasional atrophy of the intervening walls; constant accumulation of pigment cells in the modiolus and the rest of the cochlea.

2. In the spiral ganglion, the ganglion cells were diminished in number and shrunken. Empty spaces or connective-tissue new formation were found in their place in Rosenthal's canal.

3. In the smaller nerve channels, the nerve fibres were atrophic with connective-tissue new formation within the bony channels (perineuritis).

4. In the auditory nerve, atrophy and chronic neuritis were present with new formation of connective tissue in the nerve.

In chronic progressive labyrinthine deafness the changes in the hearing apparatus preponderate over those in the organ of equilibrium. The disease begins presumably in the cochlear nerve.

In a series of 1,033 new cases, thirty-one showed labyrinthine deafness; of these, twenty-four presented disturbance of hearing only, while in seven there was also disturbance of equilibrium. *Campbell.*

Deafness of the New-born; a Disturbance of the Sound-Conducting Apparatus of the Ear.

KOELLREUTTER, Rostock (*Archives of Otology,* Vol. xxxvi, No. 6). Twenty new-born children were examined in a sleeping or half-sleeping condition. There were used a musical toy, the cricri and the following forks of the Bezold-Edelmann series C, C^2 and C^3, and Galton whistle C^6.

Repeated examinations were made for several days:

1. The cricri produced a distinct reaction at all times, even a few hours after birth.

2. The tones C^6 of the Galton whistle produced a distinct reaction.
   (a) All children within the first twenty-four hours after birth.
   (b) Seventy-four per cent of all the children between two and four days old.

3. The tuning forks C, C^2 and C^3 never produced any reaction. *Campbell.*

Deafness in the Course of Acute Osteomyelitis and of Septic Processes in General.

SIEBENMANN, Basel (*Archives of Otology,* Vol. xxxvi, No. 6). To four cases published by others the author adds three which he has observed during the past five years.

It generally occurs during the first two decades in life, is usually bilateral. The first signs of deafness appeared always during the bone suppuration. The progress of the loss of hearing is usually very rapid. The two patients of Bezold and Castex, who became deaf in their seventh year, later became deaf mutes.

In all seven cases the middle ear escaped. In but one was a bacteriologic examination made and this showed the presence of a pure staphylococcic infection. In acute osteomyelitis of the long bones, this bacteriologic condition is the usual one.

Assuming that labyrinthitis and not neuritis is the cause of the osteomyelitic deafness, this is supported by the fact that both divisions of the labyrinth are involved to about the same degree. The fact that in osteomyelitic deafness occasionally marked remnants of the auditory and the static function are found, suggests the condition of the labyrinth found in meningitic deafness. Moreover, the fact that during the course of the osteomyelitic deafness no perforation from the labyrinth to the middle ear has been observed, agrees with the clinical and anatomic experiences which have been collected concerning meningeal labyrinth suppuration.

Campbell.

On the Prognosis of the Operative Opening of a Purulent Labyrinth.

FREYTAG, Breslau (*Archives of Otology,* Vol. xxxvi, No. 6). Operation on the labyrinth brings very little additional danger to the patient. The prognosis of labyrinth suppuration is improved by operative opening of the labyrinth.

Of forty-six cases observed at the Breslau Ear Clinic since Hinsberg's publication, sixteen cases of labyrinthitis were treated by exposure of the middle ear only. In these there was a mortality of 22%. In twenty-seven cases the labyrinth was opened at operation. Of these, eight cases were complicated by fatal conditions prior to operation. Of the remaining nineteen uncomplicated cases none died. *Campbell.*

A Fatal Case of Sinus Thrombosis after Chronic Purulent Otitis Complicated with Cholesteatoma, Illustrating an Unusual Variety of Infection.

KNAPP (*Archives of Otology,* Vol. xxxvi, No. 6). A boy, aged 8, suffering from kyphosis had discharge from his right ear for three years. Severe headache and vomiting existing for several months were relieved by the extraction of polypi three weeks ago. The headache returned with great severity, with vomiting, rigors and cessation of discharge. The ear-canal is collapsed, the mastoid tender and temperature 105° F. On opening the mastoid cortex it was found that the dilated antral cavity had extended into the tympanum and contained broken-down cholesteatomatous masses. There was caries of the attic and floor of the tympanum. The sinus was exposed from bulb to knee; its wall was of a yellow-black color and when incised its lumen contained disintegrated clot, thin serous pus and gas.

On the following day there was a slight chill in the morning with a temperature of 103° F. and some congestion of the optic disc. The internal jugular, facial and thyroid veins were ligated and the enlarged cervical glands removed. The bulb was further exposed and the clot removed with a curette. With each change of dressings considerable fetid pus appeared. Septic pneumonia developed and with gradually increasing cyanosis death supervened. *Campbell.*

Otalgia Considered as an Affection of the Sensory System of the Seventh Cranial Nerve.

Hunt, New York (*Archives of Otology,* Vol. xxxvi, No. 6). The ganglion of the facial nerve, the geniculate, is situated at the entrance of the aqueduct of Fallopius. Its zoster zone is to be found on the tympanic membrane, in the auditory canal, in the concha and in an adjacent marginal area of the external ear.

Otalgia is a neuralgic affection of the ear. The pain may be constant or intermittent. it may be superficial or localized in the depths of the ear. Its relative frequency to inflammatory earache is 1.8 per cent.

Reflex otalgias occur by the central and peripheral connections of the fifth, seventh, eighth, ninth and tenth nerves.

Anatomic relations exist between the facial nerve and ganglion and the trifacial nerve and its ganglion through the medium of the great and small superficial petrosal nerves.

The observation that loud and shrill sounds are accompanied by a sensation of pain in the teeth recalls the fact that the geniculate ganglion has filaments of communication with the acoustic nerve in the auditory canal.

Notwithstanding the varied nerve intercommunications, it seems clear to the author that while the facial nerve may be by no means the sole factor, it is the preponderating one in the production of otalgia. *Campbell.*

Report of Two Fatal Cases of Brain Abscess.

Thomson, New York (*Archives of Otology,* Vol. xxxvi, No. 6). Case 1. A young man, aged 18, five years previously had been operated upon for double mastoiditis. Four months radical operation was performed and this ear gave no further trouble. On the left side he has had repeated attacks of otorrhea and three months ago dizziness persisted for one day. Preceded by a hard chill, he had severe pain centering over the left mastoid scar. Profuse discharge appeared and the mastoid swelling and tenderness increased. A radical operation was performed and the dura and lateral sinus were uncovered without showing any pathologic appearance.

This operation was followed by an attack of erysipelas of the face and scalp, but the wound granulated nicely and he was free from pain.

One month later he was suddenly seized with severe pain in the head, most severe over the frontal region. There was photophobia. Temperature up to 103.5° F. and pulse to 120. There were no localizing symptoms. Leucocyte count 6800, polymorphonuclear percentage 68. There was no marked change for eleven days, then he became drowsy, the neck muscles were rigid. Leucocytes 15,000 and polymorphonuclear percentage 92. Lumbar puncture fluid was perfectly clear and showed no growth. On autopsy a large cerebellar abscess was found which involved the entire left lobe, a portion of the central lobe and encroached on the right lobe. Its walls were indistinct and it did not show any capsule formation.

Case 2. A man, aged 50, who complained only of deafness in the left ear which was of two weeks' duration. On examination the left auditory canal was so swollen that the mt. could not be seen. There was no tenderness whatsoever, yet the anterior and posterior canal walls were incised without result.

Two weeks later he complained of dull nocturnal pain on the left side of the head followed in another fortnight by painful swelling over the root of the zygoma. On making an incision there pus was evacuated.

Two months after coming under observation, his mastoid and zygomatic region became intensely swollen, red and tender. On operation the mastoid cells were disintegrated and filled with pus. The dura above the antrum and the sinus were covered by granulations which were left undisturbed as there were no intracranial symptoms. Next day he was somnolent and two days later aphasia developed. This was followed by paralysis of the right side of the face and right arm, scanning speech and left optic neuritis. The temporo-sphenoidal lobe was explored through the mastoid wound and about one dram of pus evacuated. His condition grew gradually worse and coma preceded death.

On autopsy an abscess cavity about the size of an English walnut was found in the temporo-sphenoidal lobe filled with thick creamy pus. *Campbell.*

A Case of Acute Labyrinthitis Due to Meningitis.

YEARSLEY, London (*Archives of Otology,* Vol. xxxvi, No. 5). A man, aged 24, who at the age of 1½ years had a serious illness during which time he put his hands to his head frequently and was continually shaking it, so that evidently he had severe head pain. He made no attempt to speak until between the age of 3 and 4 years.

Weber's test lateralized to the left:

Loud voice R = 0
L = 9 in.
Bone conduction C Fork R = 0
Left = — 32 sec.
Upper tone limit Edelman whistle R = 0
L = 5.6

The retention of the lower tone limit is due to the fact that the middle ear is intact. *Campbell.*

Intratympanic Operations Under Local Anesthesia.

MILTON J. BALLIN (*New York Medical Journal,* October 5, 1907), after referring to the failure of applications of solution of cocain to fully deaden the drum, recommends in cases requiring paracentesis where a general anesthetic is not indicated or allowed, the use of a weak solution of cocain introduced into the mucous membrane of the auditory canal in the following manner: "The external auditory canal is first thoroughly cleansed with any antiseptic solution. A small piece of cotton is then placed into the canal so as to protect the drum against the ethyl chlorid spray, which would otherwise cause too much pain. Having done this, a cleft speculum is introduced in such a manner that only the superior wall of the canal is exposed to view. With the aid of an assistant a spray of the ethyl chlorid is now allowed to play on this exposed surface, and as soon as the parts are well anesthetized the needle of the syringe is quickly introduced at the junction of the bone and the fluid slowly injected as already stated; the writer uses for this purpose 10 to 15 minims of a 1 per cent solution of cocain to which 5 to 6 minims of adrenalin solution (1-1000) have been added. The injection must be carried out very slowly so that infiltration takes place gradually, thus avoiding unnecessary pain. Having done this, little piece of cotton is then removed from the canal. We now wait for four to five minutes, at the end of which time it will be noticed that the drum has assumed a more or less bleached appearance, and has also become so insensible to pain that paracentesis can be performed without any annoyance to the patient.

Harris.

The Radical Mastoid Operation.

FRANK ALLPORT, Chicago, Ill. (*Jour. A. M. A.*, March 30 and April 6, 1907), here gives in great detail and with many excellent illustrations, the complete technic of the radical mastoid operation as performed by him. He regards the electric engine and rotary burrs most useful additions to the armamentarium. *Richards.*

Some Important Surgical Relations of the Temporal Bone.

GEORGE E. SHAMBAUGH, Chicago, Ill. (*Jour. A. M. A.*, 1907). The sigmoid sinus, the facial nerve, the lateral semicircular canal, the external carotid, and the jugular bulb, should be familiar to the operator not only as to their relation to the tympanic cavity and mastoid cells, but he should also be familiar with their variations. At the tympanic orifice of the tuba auditiva the carotid is separated from the tube by a thin shell of bone which can readily be penetrated by the curette. The force of the curette can be applied with safety only along the external or anterior wall of the tube, because along its internal wall lies the canal of the carotid.

Richards.

The Treatment of Chronic Suppuration of the Middle Ear Without Removal of the Drum Membrane and Ossicles. The Duty of Restoring Hearing by Operation in Chronic Aural Suppuration.

CHARLES J. HEATH, London (*London Lancet*, April 27, 1907). Address before the West Kent Medico-Chirurgical Society, May 3, 1907. These papers are supplementary to the original paper published in the *London Lancet*, August 11, 1906. The author reports some more cases and gives the experience of other aural surgeons tending to prove his claims that the operation for chronic suppuration as performed by him conserves the hearing whereas after the radical operation or the operation of ossiculectomy, the hearing may be and frequently is worse, as the operation is done solely to stop the discharge and not at all to save the hearing. Mr. Heath considers the antrum to be the key to the position in nearly every case of suppurative ear disease and thinks if the disease in that cavity is cured that the morbid changes in the attic and tympanum will spontaneously disappear. This operation is not a simple opening up of the antrum and a clearing of that cavity and the aditus followed by drainage, as in Schwartze's operation, which caused so many disappointments and led to the institution of the radical operation, which, on the other hand, went much too far. In the new operation not only the posterior superior part of the meatus is removed, as in some other operations, but the antrum and aditus are practically eliminated, as well as all disease associated with those cavities. The whole posterior wall of the bony meatus and even its arched floor must also be removed, and this latter proceeding is followed in no other operation, but it must be done if that full access to the tympanum which is necessary both during and after the operation is to be assured. This is essential not only for the safe and thorough removal of granulations, polypi, etc., at the operation, but especially for the dressings during the after-treatment, for without the removal of the floor it is impossible to see the whole of the membrane; moreover, a pocket would be left between the arched floor and the membrane where discharge might accumulate. *Richards.*

A Restudy of the Minute Anatomy of Structures in the Cochlea with Conclusions Bearing on the Solution of the Problem of Tone Perception.

GEORGE E. SHAMBAUGH, Chicago (*The American Journal of Anatomy*, August 1, 1907). As the results of his anatomic studies the author has formed a new "resonator theory" of tone perception in which he considers the vibrating mechanism of the membrana tectoria as the seat instead of the membrana basilaris as was supposed by Helmholtz. Throughout the entire length of the cochlea the mechanism of the long rows of hair cells is admirably suited to the requirements of the "resonator theory." "These hair cells are the real end-organs, wherein the transference of the physical sound waves to nerve impulses takes place. Each cell, or rather group of cells, when adequately stimulated, leads to the perception of a particular tone. The higher tones, presumably, are taken up by the cells located in the basal coil, the lower tones by the cells near the apex of the cochlea." It is necessary to have some vibrating body to set in motion the hair cells of the organ of Corti, and for this purpose the author thinks the rigid membrana basilaris can not be the vibrating structure which it was believed to be by Helmholtz. He traced the basilar membrana through carefully made sections near the beginning of the vestibule and found that this membrane at a considerable distance from the point where the cochlea tube begins, became so thick and rigid as to preclude any idea of its being a vibrating structure. This membrane also disappears at the point in the lower end of the basal coil where the organ of Corti is still perfectly formed and this fact points to the conclusion that the vibration of this membrane is not essential for hearing. Seeking now to ascertain how impulses are transferred to the projecting hairs the question arises, can these hairs be stimulated directly by impulses in the endolymph without the intervention of the membrana tectoria? After investigating the character and relations of this most interesting membrane the author became convinced that the tectorial membrane does not float free in the endolymph above the cells forming the organ of Corti. This membrane on account of the distortion produced by the fixing and hardening agents necessary in preparing the labyrinth for histologic study has been much distorted as is shown by the various drawings which have appeared in the published works on the labyrinth. The author is certain that this membrane does not float free in the endolymph above the organ of Corti but that this position shows the one usually described as the result of the shrinking of the structure by the fixing agents. He gives a number of drawings of different sections all drawn to the same scale, showing that the hairs of the hair cells actually project into the under surface of the tectorial membrane and are not merely brought into touch with it by accident.

The membrana tectoria when examined throughout the several coils of the cochlea shows great variations in size from one end to the other, being many hundred times larger near the apex of the cochlea than at the beginning of the basal coil and this increase in size is gradual. The membrane possesses a lamellar structure. This lamellar structure gives the membrana tectoria somewhat the appearance of a soft feather and the lamellae vary in length with the varying size of the tectorial membrane, the shorter lamellae occurring near the beginning of the basil coil and the longest at the apex of the cochlea. They are supported and held together by an apparently homogeneous semi-fluid substance, the specific gravity of which appears to be the same as that of the endolymph in which the membrane is suspended. This is shown by the fact that in spite of its semi-fluid character it sustains its position in the endolymph though supported only by its contact with the labium vestibulare and where the 'Streifen of Hensen' is in contact with the supporting cells of the organ of Corti." These characteristics of the membrana tectoria render it suitable to respond to the most delicate impulses passing through the endolymph. The great variation in size of the membrane from one end of the cochlea to the other, together with its lamellar structure, are physical characteristics which suggest the probable basis for a series of resonators which make it possible for the membrane in one part of the cochlea to respond to impulses of a certain pitch and in another part to impulses of another pitch. In this way the small impulses produced by the high pitched tones would in some way set to vibrating the tiny tectorial membrane found in the beginning of the basal coil, while the larger impulses arising from the deeper tones would produce vibrations in the much larger tectorial membrane found in the upper coils.

The vibrations in a part of the membrana tectoria produced by a particular tone must necessarily involve a considerable area of this structure. As a result a more or less extensive group of hair cells is stimulated. The nerve impulses arising from the stimulation of the several hair cells included in this group come together in the brain center of the cortex, where the tone picture forms the final step in the perception of this particular tone. When a tone slightly higher or lower than this one is produced, the same group of hair cells is stimulated, excepting for the addition of a few more cells at one end and the loss of a few cells at the other end of the area involved. The sum total of the impulses which reach the center in the brain is, therefore, different for every tone, however near they may be in the scale. The ability to distinguish between the several tones is, therefore, a matter of education, since anatomically we encounter no difficulty in accounting for a different tone picture for each particular tone difference.

A necessary corollary of the fact that the vibration of the membrana tectoria resulting from a particular tone spreads over a considerable area of this membrane is the fact that when two tones near each other in the scale are produced simultaneously the two areas of the membrana tectoria thrown into vibration will overlap to a greater or less extent. This overlapping of the areas stimulated by several tones was first conceived by Helmholtz in his basilar membrane theory and has been looked upon by many as one of the strongest arguments in favor of his resonator hypothesis since it offers perhaps the most plausible explanation of the most difficult of all the secondary phenomena of tone perception to be accounted for, namely the phenomenon of "beats." The objections to this ingenious conception of Helmholtz which are based on the very apparently rigid inflexible character of the membrana basilaris do not hold when the delicate film-like, semi-fluid membrana tectoria takes the role of resonator.

Again, the peculiar alterations in the function of hearing which occur as the result of pathologic changes in the cochlea, find a more ready explanation on the hypothesis that the membrana tectoria is the resonator than that the membrana basilaris fills this role.

In the first place, the occurrence of "tone islands," that is, the preservation of certain circumscribed areas of hearing in cases where there has been more or less extensive destruction of hearing, finds as ready an explanation with the membrana tectoria as resonator as when we attribute this function to the membrana basilaris.

In the second place, the explanation of the pathologic phenomenon known as "diplacousis binauralis dysharmonica," where the patient hears a tone in the affected ear of a pitch different from that heard in the normal ear, is much more readily accounted for on my hypothesis. This peculiar phenomenon is readily explained as due to some slight alteration in the delicate and easily disturbed membrana tectoria which causes it to respond over a certain area to impulses of a different pitch than when in the normal condition.

Finally, an explanation of "tinnitus aurium" or subjective noises may possibly be found in this conception of the structure and function of the membrana tectoria. In the first place, the character of tinnitus aurium is usually that of an indefinite sound like the wind in the forest or the rushing of water, sounds made up of a great complexity of tones and with no definite pitch. Clinically, these subjective sounds arise from a variety of pathologic conditions. One of the best known causes of tinnitus is pressure applied to the conducting apparatus so as to push the foot plate of the stapes into the oval window. This results in tinnitus aurium of the indefinite character described above. What actually takes place when the stapes is thus forced into the oval window is an increase in the pressure of the intralabyrinthine fluid. The result of this alteration in pressure must be a disturbance of the membrana tectoria which has apparently the same specific gravity as the endolymph when the latter is under its normal pressure. The hairs from the hair cells, as I have shown above, normally penetrate into the lower surface of the membrana tectoria. Any disturbance in this membrane, however slight, would, therefore, alter the normal relations existing between the membrane and the hair cells. It seems that such an alteration from the normal relation between membrana tectoria and the hairs of the hair cells would constitute a stimulation of these cells. When the foot plate of the stapes is pushed into the oval window there would result a slight stimulation of perhaps all the hair cells in the cochlea. The result would be exactly what we meet with clinically, a tinnitus aurium of an indefinite character like the wind in the forest or the roar of a sea-shell. When a sudden increase or decrease in the blood-pressure results in tinnitus aurium the cause is the same as when the stapes is pushed into the oval window. The explanation for the increase or decrease of the intralabyrinthine pressure is here quite evident. The tinnitus aurium arising from the administration of certain drugs is also plausibly explained in the same way as due to an alteration in the blood-supply to the labyrinth with resulting alteration in the pressure of the intralabyrinthine fluid. The tinnitus occurring in Meniere's disease, where there has been an escape of blood into the cochlea, is also similarly accounted for by this conception of the physiology of tone perception. The disturbances in the function of hearing arising from an injury produced by a shrill whistle or an explosion near the ear are also readily explained. In the first place, when a permanent disturbance in hearing is thus produced it can be readily accounted for by a partial severance of the relation between the membrana tectoria and hair cells so that the hairs from a greater or smaller number of these cells project free in the endolymph and do not come in contact with the membrana tectoria and, therefore, can not receive the stimulation from impulses passing through the endolymph. On the other hand, when there results from such an injury a permanent tinnitus aurium, this is explained by a partial, not complete, severance of the membrana tectoria from the hair cells over a certain area. This alteration of the relation existing normally between the hair cells and membrana tectoria may result, as we have repeatedly pointed out, in a stimulation of these cells. This explanation appears all the more rational since the pitch of the tinnitus is often approximately that of the whistle which originally produced the injury.

"To restate briefly the process by which the phenomenon of tone perception is accomplished: The sound waves conducted from the air impinge upon the membrana tympani, producing vibrations in it. These vibrations conducted along the chain of ossicles transmit impulses to the intralabyrinthine fluid through the medium of the foot-plate of the stapes. The impulses originating in the fluid in the vestibule pass directly into the scala vestibuli and through the membrane of Reissner to the endolymph where sympathetic vibrations are imparted to the several parts of the membrana tectoria depending on the pitch of the tone. The vibrations of the membrana tectoria in turn stimulate the hairs of the hair cells which normally project into its under surface. The nerve impulses originating from all the hair cells thus stimulated by a particular tone come together in the brain center in the cortex when the tone picture forms the final step in the process of tone perception."

Richards.

II.—NOSE.

Primary Nasal Diphtheria.

GEO. STIMSON (*New York Medical Journal,* December 14, 1907). After a report of several cases, one in an infant ten days old, Stimson proceeds to describe the symptoms and course of the disease and draws the following conclusions: "Primary nasal diphtheria is a more common disease than is ordinarily supposed." It is usually a benign affection, the constitutional symptoms being not marked or even entirely absent

The primary form is very seldom malignant per se, and especially is this true if it remains confined exclusively to the nares. It displays a tendency to limit itself to the nose. When it does spread to portions of the respiratory tract lower down, the severity, that is, the toxemia and systemic disturbance, seems to increase pari pasu with, and as a direct result of, such extension. There is a decided tendency towards chronicity. The prognosis is, as a rule, good. Paralytic phenomena, such as loss of knee jerks, paralysis of the palate, etc., are extremely rare."

It is capable of acting as a focus of infection, and the resulting cases may be of a malignant type. The importance of early detection of this disease by bacteriologic examinations of all cases of persistent purulent rhinitis of doubtful origin, especially in children, cannot, therefore, be overestimated.

Harris.

A Nasal Sporozoon.

JONATHAN WRIGHT (*New York Medical Journal*, December 21, 1907), reports the discovery of a nasal sporozoon—rhinosporidium Kinealy—in a specimen of benign warty growths removed from the nose of a farmer living in Tennessee. He identifies this with what has already been described by Kinealy and Beattie: "In all cases observed the organism has occurred in granulation tissue near the vestibule of the nose, which suggests the inference that the infection was carried by the finger nail. Wright describes the sporozoa as a group of exclusively parasitic protozoa. They always possess the power of reproducing endogenous minute protoplastic nucleated corpuscle or sporules, a part of their own bodies. These are protected by a cyst envelope resistant for a time to the external environment, and without this the sporules are not reproduced or do not continue their development. He shows the distinguishing features of this particular form of sporozoon and illustrates them by several microphotographs.

Harris.

The Questionable Influence of So-called Diathetic Condition in Diseases of Throat and Nose in Children.

CHARLES GILMORE KERLEY (*New York Medical Journal*, August 24, 1907) does not believe in the so-called diathetic influence on diseases of the nose and throat and concludes as follows:

"To my mind the three factors, the sucking-in habit, the free use of cane sugar, inducing colds, and the tendency to lymphoid tissue involvement, a condition peculiar to all children, contribute largely to the production of the adenoids."

Harris.

Intracranial Complications of Diseases of the Accessory Sinuses of the Nose.

LEWIS A. COFFIN, New York, (*N. Y. Med Rec.,* November 9, 1907). Three cases coming under the author's own observation are reported and a review of the literature of the subject given. He thinks that the increase in the mortality from accessory sinus suppuration has been more apparent than real, since at present the diagnosis of disease of these cavities is made much more frequently than formerly. Operations on these sinuses should only be done by operators who are willing to sufficiently educate and prepare themselves for the work. He quotes Prof. Killian as stating that out of 100 operations on frontal, ethmoidal, and sphenoidal sinuses he had had only three deaths.

Richards.

Glottic Spasm of Nasal Origin in Adults.

FRANK A. CONLON (*Boston Med. and Sur. Journal,* August 8, 1907). The author reports a case of his own and gives histories of fifteen other reported cases. In his own case, removal of the middle turbinate stopped the attacks which were very severe. The reflex travels along the trigeminus and toward the periphery in the vagus. *Richards.*

The Indications for Resection of the Middle Turbinate.

W. E. CASSELBERRY, Chicago, Ill. (*Jour. A. M. A.,* July 6, 1907). The middle turbinate is resected for the following reasons:

"To promote drainage and counter-drainage in nasal accessory sinus suppuration and for access in diagnosis and treatment.

To promote drainage and cleansing in certain types of atrophic rhinitis (atrophic ethmo-rhinitis).

To relieve edematous turgescence and to provide access for the radical treatment of non-suppurative ethmoiditis and nasal polypus.

To suspend pressure leading to headache, neuralgia, eye symptoms and broadening of the nasal bridge, etc.

To improve nasal respiration and ventilation.

To relieve hyperesthesia and to diminish certain reflexes, e. g., sneezing, asthmatic stimuli," etc. *Richards.*

III.—LARYNX.

Acute Edematous Eversion of the Ventricle of Morgagni.

T. J. REARDON (*New York Medical Journal,* June 29, 1907). Eversion of the ventricle is an exceedingly rare condition. Edema of the ventricle may accompany edema of the epiglottis or of the aryepiglottic folds. Reardon reports a case in a singer, age 40. A few days previous to the attack, she had suffered from what was diagnosed as acute follicular tonsillitis. After singing in church she became hoarse and the following day was cyanotic and dyspneic. Her voice was a hoarse whisper. Laryngoscopic examination showed the right ventricle of Morgagni everted and so edematous as to render a view of the vocal cords impossible, the mass being as large as a pigeon's egg. Intubation was performed and gave immediate relief. Twenty-four hours later the tube was removed and the larynx was found normal. The author discusses the pathology and treatment of edema and decides in favor of intubation over other measures though advising that a tracheotomy set be kept at hand. *Harris.*

Concerning the Laryngospasm of Adults.

LUDWIG NEUFELD (*Archiv. für Laryngologie und Rhinolgie,* Bd. xx, Heft, ii, 1907). Laryngospasm in adults is much rarer than in children. The etiology of this condition is still very obscure, although hysteria and neurasthenia have been given as fairly frequent causes. Organic changes in the neck and larynx particularly tumors, may bring it about.

The author reports the following case: The patient, an unusually robust man, had been subject for six weeks to severe suffocative attacks, coming on once or twice every day. The slightest desire to cough brought on laryngospasm. The patient's five children were down with whooping cough. On examination nothing abnormal was found in the throat, the larynx appearing perfectly normal. Inhalations had a good effect and after six weeks the attacks gradually lessened.

Another case reported by the author belongs to the class of cases described as ictus laryngis. The etiology of this class of cases is also very obscure. The patient, a blacksmith aged 23 years, had always been well up to a year before. At that time he slept in a room in which the stove smoked a good deal. He developed a severe laryngitis which was treated. From that time until seen by the writer he had had severe attacks of suffocation.

On examination, the vocal cords were found slightly reddened but otherwise the larynx was normal. Suddenly during the examination the cords closed firmly and immediately the patient fell to the floor. Cyanosis was marked, and there was an inspiratory stridor.

The attacks lasted twenty to thirty seconds, when the cyanosis gradually disappeared, and was followed by an intense pallor, with a slow pulse and dilated pupils which did not respond to light.

Such attacks occurred as often as four times a day. They could be prevented by pressure against the larynx, but came on when he laughed, or became excited. The author believes this case belongs in the class of traumatic neuroses.

Theisen.

IV.—PHARYNX.

Late Secondary Hemorrhage, Following the Removal of Adenoids.

F. L. WACHENHEIM (*New York Medical Journal*, October 26, 1907), reports two cases of profuse hemorrhage following the adenoid operation. In each instance it occurred at an interval of five days. He condemns the use of forceps as tending to leave residua and thus cause bleeding.

Harris.

Post-Operative Treatment of Adenoid Patients.

BRYAN DE F. SHEEDY, New York (*American Journal of Surgery*, March, 1908). Children must be educated in the matter of proper nasal respiration and should be put through a course of thorough gymnastics lasting over a period of one to five years. Deep nasal breathing with the mouth closed, for a few minutes at a time, eight to ten times a day, will do much to develop air space in patients who have been mouth breathers. Expiration as well as inspiration must be done through the nose. The lips must be kept closed during sleep by strips of adhesive plaster placed over the mouth at bed time. *Richards.*

V.—MISCELLANEOUS.

Ligation of the External Carotid in Rhinology and Pharyngology.

CHEVALIER JACKSON (*New York Medical Journal,* August 17, 1907) has ligated the external carotid artery thirty-eight times in thirty cases, both arteries being tied in eight cases; to arrest hemorrhage after tonsillectomy, seven cases; to forestall hemorrhage in extirpation of malignant disease of the tonsil and tongue, four cases; to forestall hemorrhage in extirpation of malignant disease of the maxillary antrum, three cases; to inhibit growth in malignant disease of the maxillary antrum, four cases; to forestall hemorrhage in extirpation of nasopharyngeal fibromata, eight cases; to arrest spontaneous nasal hemorrhage, two cases; to arrest postoperative nasal hemorrhage, two cases. *Harris.*

The Etiology of Tuberculosis of the Upper Respiratory Tract.

G. B. WOOD (*Laryngoscope,* December, 1907) states that in a certain per cent of persons undoubtedly virulent tubercle bacilli will be found in the nasal cavity, but it is not possible that they remain there for any length of time. Knight states that up to 1901 there had been 108 cases of tuberculosis of the nose reported. It is very likely that a large number of cases of tuberculosis of the nose are overlooked, but even then it is remarkable that so few cases had been reported. The author, who has had an extended experience in examining the nasal cavities of cases of pulmonary tuberculosis in the Phipps' Institute, has never seen a case of primary nasal tuberculosis. It would seem that the nasal cavities are only rarely infected with the tubercle bacillus, probably because the time necessary for the propagation of this slow-growing organism permits the cilia of the nose and the nasal secretions to remove it from the fossae; but it is also very likely that the nasal secretions themselves are directly inhibitory to its growth.

Tuberculosis of the pharynx should be considered as practically identical with tuberculosis of its lymphoid tissue. When the comparative susceptibility of the various parts of the upper air passages to tuberculosis is considered, preference for liability to infection must be given to the tonsillar tissues. The results of all the research work done in relation to general microbic invasion through the tonsils, show, that in the tonsils more than anywhere else on the body surface, the penetration of the invading germ depends not so much upon any mechanical barrier presented by the tissue as upon the relative virulence of the germ.

The author, quoting from a paper published by himself in 1904, states, that the tonsils of 136 cases of pulmonary tuberculosis were examined by seven observers, and in ninety-four, tuberculous involvement was detected. The diagnosis in these cases was made mainly by histologic examination. In very advanced cases of pulmonary tuberculosis the tonsils almost never escaped infection. In nine cases reported in 1904 by the author, in which postmortem examinations were made, the tonsils in every one showed tubercles with giant cells, and in thirty-eight cases examined during the past year, he found histologic changes typical of tuberculosis in thirty-five. In a series of 1,671 cases of hypertrophied tonsils compiled from the published records of twenty-three authors, primary tonsillar tuberculosis was diagnosed in eighty-eight or 5.2 per cent. The author found the same proportion in entirely unsuspected cases when the tonsils had been removed by operation. It would seem safe to assume that about 5 per cent of children had tuberculosis of the tonsils.

The author then discusses the method of infection in pulmonary tuberculosis, particularly the liability to systemic infection from tuberculous lesions in the tonsils. If a given part of the body is inoculated, the lesion develops locally, and is generally propagated through the lymphatics of the nearest set of lymph nodes. The bacillus is arrested in these nodes, and cannot proceed further until destruction of this barrier has been accomplished.

The tonsils may be described as differentiated lymph glands, but it is not probable that they possess the same filtering qualities as the lymph nodes.

The writer has been able by rubbing a virulent culture of bovine tubercle bacilli on the surface of the tonsils of hogs, not only to inoculate the tonsils, but succeeded in finding the tubercle bacillus in the regionary lymph gland of the neck within five days after the inoculation. In the same series of experiments he found that histologic changes appeared as early in the cervical lymph nodes as in the tonsil. In summing up, he states that the clinical importance of a tuberculous lesion in the tonsillar tissues of the throat is about the same as a local tuberculous lesion in any other non-vital part of the body which is separated from the internal organs by an intervening chain of lymph nodes. *Theisen.*

Gastroscopy.

CHEVALIER JACKSON, Pittsburg, Pa. (*Medical Record,* April 6, 1907). Appearances of the stomach shown through the gastroscope are described and eight cases reported showing the value of the gastroscope as a diagnostic agent. "Gastroscopy is not simply a feat. It has a field of usefulness that will increase as our skill and knowledge increases. Gastroscopy, in my opinion, is useful for the detection and removal of foreign bodies, the diagnosis of many pathologic conditions, as inflammation, ulceration, scars, neoplasms, dilated vessels, and at least one of these conditions, namely, benign ulcer. When the gastroscope shall have gained its deserved recognition, malignant disease of the stomach will be diagnosticated in many instances sufficiently early to give the abdominal surgeon a fair chance. Better still, a positive diagnosis of precancerous conditions will be made sufficiently early to enable him to save lives that are now being lost through reluctance of the patient to submit to an exploratory celiotomy. Gastroscopy is not offered as a substitute for an exploratory operation in every instance, but only in those cases in which the patient declines to submit, or in which the surgeon or the physician considers a simple procedure almost free from risk preferable to operative exploration.

Gastroscopy is the only method known today that yields ocular information as to the condition of the gastric mucosa. Other methods give information only by inference from the disturbance of function. Thus gastroscopy becomes an auxiliary." *Richards.*

The Oral Cavity as a Factor in the Development of Speech.

G. HUDSON-MAKUEN, Philadelphia, Pa. Normal nasal breathing cannot always be brought about by rendering the nostrils patent or by the removal of adenoids, but in addition to these measures, deformity of the teeth and maxillary arches must be corrected so as to allow the natural closure of the mouth and conserve the tonicity of the facial and buccal muscles. Hare-lip and cleft palate should be relieved at once, the effect on the speech being much better the earlier it is done.

Richards.

ANNALS

OF

OTOLOGY, RHINOLOGY

AND

LARYNGOLOGY.

VOL. XVII. JUNE, 1908. No. 2.

IX.

SURGERY OF THE FACIAL NERVE.

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Chicago.

Aside from the report of five cases of facial hypoglossal anastomosis for the cure of permanent facial paralysis that I have performed, and the demonstration of a gross specimen removed postmortem from one of these cases which had been cured for over a year, showing the perfect union of the two nerves, I have nothing original to offer in this line of work. Consequently the large part of my article is a repetition of what is already well known and on record. I have, however, written up this subject with one special purpose in view, namely: Inasmuch as the greatest number of facial paralyses are due to pathologic conditions within the temporal bone, more especially in the field of the otologist, or following his operative procedures to eradicate it, as, for instance, the radical mastoid, jugular bulb and labyrinthine operations, I have decided to bring this subject before you *in extenso*, compiling all that is practically known of the anatomy, physiology, pathology and treatment of the facial nerve.

Up to the present time, this subject is considered more particularly in general surgery, neurology, and internal medicine, but it evidently falls within the realm of the modern otologist.

It becomes frequently necessary to do a radical mastoid operation, in order to cure a facial paralysis, or if following such an operation one obtains a permanent facial paralysis, there is every reason why the otologist should correct this condition and not depend on a general surgeon to do so.

While the operation is very delicate and tedious, it is not difficult, and certainly the delicacy and tediousness of any operation are more characteristic of the work of the otologist than that of the general surgeon.

The operations for the cure of facial paralysis have gained considerable favor in the past five years, so that at the present time we have a great deal of material at our disposal to study the results from various methods. Murphy's article on surgery of the nervous system compiles practically all the literature up to the present day, comprising thirty-four cases, and completely goes over the pathology of the peripheral nerves. I am, through the kindness of Dr. J. B. Murphy, using that part of his article practically verbatim.

In order to study the cure of facial paralysis, one should have a clear knowledge of the anatomy and function of the facial nerve, with all its associated branches. I have, therefore, prepared these cuts, which will also serve to show the relationship in studying the anastomosis of the various nerves with the facial. Figure 1 shows the course of the facial nerve from the beginning at the cortex (upper segment), traversing the internal capsule and associating or uniting with the nucleus of the facial nerve at the floor of the fourth ventricle. From thence we speak of the facial nerve proper, or the lower segment. The fibers, then, pass in a curve about the nucleus of the sixth nerve, making what is known as the knee, being then joined by the eighth or auditory nerve, as two distinct trunks. These nerves have their exit from the brain at the junction of the pons and medulla. A third little nerve, known as the nerve of Wrisberg, or nervus intermediaris, is seen between these two trunks in their course towards the internal auditory meatus. At this latter point the two nerves are in a distinct relation; the auditory is posterior to the facial. They then pass into the internal auditory meatus until the aqueductus Fallopii is reached, when the facial leaves the auditory nerve and passes into this (Fallopian canal) across the roof of the middle ear cavity. Then it takes a turn backwards and downwards, so as to occupy the innermost part of the lower half of the posterior meatal wall; it then passes through the bony canal in the mastoid process until it reaches the stylo-mastoid foramen. Here it again takes a horizontal turn towards the posterior surface of the parotid gland, just below the cartilaginous external auditory meatus, piercing the capsule of this gland, and entering the substance of it.

The nerve then divides at first into two branches, one of these again giving off a third. These three branches leave the gland, giving off a number of small branches, which pass to their respective muscles, anastomosing with the sensory nerve of the face, forehead and neck.

The various branches as they are given off, also branches associating or communicating with other nerves, are as follows:

Interosseous Branches:

1. Great superficial petrosal nerve, which communicates with the fifth.
2. Anastomotic branch with tympanic plexus.
3. Stapedius nerve.
4. Chorda tympani.

After Leaving Foramen:

1. Posterior auricular nerve.
2. Digastricus nerve.
3. Stylohyoideus nerve.
4. Communicating with the glosso-pharyngeal.

Parotid Plexus:

1. Ramus coli.
2. Mandibular ramus.
3. Buccal ramus.
4. Zygomatic ramus.
5. Temporal ramus.

FUNCTION OF THE FACIAL NERVE.

The facial is the motor nerve of all the muscles of the face, including the platysma, but not including any of the muscles of mastication. It supplies the parotid gland, and through its communication by means of the petrosal with the vidian, some of the muscles of the soft palate, probably the levator palati and azygos uvula (denied by some). By its tympanic branches it supplies the stapedius; through the otic ganglion the tensor tympani; through the chorda tympani it sends branches to the submaxillary glands, and to some muscle fibers of the tongue, through its connection with the lingualis. It supplies the mucous membrane of the anterior two-thirds of the tongue. By some of its branches before it comes upon the face, it supplies the muscles of the external ear, the posterior belly of the digastric, and the stylohyoideus. It, therefore, is a secretory nerve, as well as a nerve of special sense, aside from its motor functions. The sense of hearing is also affected by the muscles which it suplies, as the stapedius and tensor tympani. Again, the sense of smell is somewhat controlled by this nerve, in that it permits proper dilatation of the alae nasi; in this manner a proper amount of air is inspired. The sense of taste is supplied by the chorda tympani to the anterior two-thirds of the tongue. Some observers claim that this branch is an entirely independent nerve, only accompanying the facial, but having its origin from the nucleus of the fifth. Sight is influenced by its action on the lids.

The surgical anatomy will be described in connection with the operations.

CAUSES OF FACIAL PARALYSIS.

These will depend a great deal on the different portions of the nerve involved; for instance, the intracranial, the interosseous, or the external portion. However, one may have a paralysis due to the involvement of two of the portions, as an ascending or descending neuritis. Enumerating some of the most frequent causes we have, first, lesions, as:

1. Hemorrhages, subcortical, in the internal capsule, and before the communication with the nucleus.
2. Brain abscess.
3. Gumma.
4. Tumor.
5. Meningitis.
6. Suppurating disease of the middle ear, the most frequent cause, in the interosseous portion. It is most frequently in the region of the Fallopian canal, although it may occur anywhere along its course. Frequently it is associated with Bezold's mastoid abscess.
7. Traumatic, as following the mastoid operation (radical),

or labyrinthine exsection It sometimes follows pressure from gauze packing, or improper manipulation of the protectors for the facial nerve. Fractures of the base of the skull are at times followed by facial paralysis, or gunshot wounds of the temporal bone.

In the external portion we find it associated with:

8. Tumors of the neck.
9. Retromaxillary tumors or abscesses.
10. Following resection of glands of the neck.
11. Osteoperiostitis of the lower jaw.
12. Tumors and abscesses in the parotid gland, and operations for these conditions.
13. Trauma, as stab wounds or the characteristic gashes of the German duelists, causes a goodly number of external facial paralyses.

The general systemic diseases that cause facial paralysis are:

14. Syphilis.
15. Rheumatism.
16. Other acute infectious diseases.

Among the largest group of cases are those spoken of as Bell's palsy, which are supposed to be due to:

17. Exposure to cold and draught.
18. Toxic.

According to others:

19. A severe gastro-intestinal toxemia may cause facial paralysis.

Such local causes as:

20. Very strong remedies poured into the ear, or applied, as chromic acid, trichloracetic acid, nitrate of silver, have been reported as causing paralysis.
21. Excessively hot or cold instillations into the ear with perforating drum membranes are given as causes of paralysis.

HISTOLOGY OF THE FACIAL NERVE.

A cross-section of the trunk of the nerve shows, as any other peripheral nerve, the following structures: (Fig. 2.)

Section (transverse) of the nerve trunk shows in the first place bundles of nerve fibres (funiculi) ensheathed by a fibrous capsule (epineurium). Each bundle is surrounded by its own sheath known as the perineurium. Each nerve fibre is surrounded by a sheath known as endoneurium. Between the structures are seen lymph spaces, veins, arteries, fat.

The termination of the nerve fibres is by the muscle plates, which is the axis-cylinders breaking up into a number of cells, which are practically fused with the sarcolemma of the muscle.

Section of Nerve Fibres (fig. 3):

(1) External (primitive nerve sheath) nucleated sheath of Schwann, or neurilemma.

(2) Intermediate or packing substance, also known as medullary or myeline sheath, or white substance of Schwann.

(3) Internally the axis-cylinder, otherwise known as primitive band, axis band, or axial fibre.

The neurilemma is structureless, and within it are seen at intervals nuclei, surrounded by protoplasm.

The medullary substance is supposed to be made up of two distinct parts: (a) a reticular framework, (b) fatty substance of a semi-fluid consistency, staining readily with osmic acid. The reticular network, according to some authors, has a highly chromatic property which is very resistant to chemical and digestive fluids, known as the neuro-keratin network.

The axis-cylinder consists of primitive fibrillae and can be traced from origin to termination.

Nodes of Ranvier. At regular intervals in the nucleated sheath of Schwann one can see a constriction. At these points the external sheath and axis-cylinder come in immediate contact. Between each two nodes of Ranvier is what is termed an internode. The latter varies in size, but averages about 1 mm. Only one nerve nucleus to each internode can be demonstrated. The constricting band of Ranvier is found between two internodes, uniting them to the external sheath, which band stains black with silver nitrate. Since the axis-cylinder also stains with silver nitrate, one can therefore at each node of Ranvier see a cross known as cross of Ranvier.

The size of a nerve fibre varies much in its course, the average being 6.2 to 8.7 micra.

PATHOLOGY OF THE FACIAL NERVE.

We may have either (a) neuritis or (b) death of the nerve. In the case of the former there is an increase in the vascular and plasmodic supply, with swelling of the component parts of the perineurium. There occur a round-cell infiltration and naturally a compression of the axis-cylinder. The degree of inflammation varies and in the marked cases may terminate in permanent destruction of the nerve, that is, there may be gangrene or suppuration.

In the case of severance of the continuity of structure of the nerve, either by being cut or injured, as in fractures or gunshot wounds, in association with and complication of a suppurative process of the temporal bone, or tumors involving the nerve cell, one will see the following changes: According to F. Marchand,[1] the whole peripheral end of the nerve degenerates, as well as a small part of the central, up to the first node of Ranvier. On the second day after the division, the medullary sheath breaks down in the form of cylindrical fragments, which continue subdividing during the next few days. These fragments are still nucleated in the sheath of Schwann. The axis-cylinders also fragment, and are either swollen or fibrillary in aspect. They are included among the myelin fragments. At the same time a regenerative process begins in the nuclei of the sheath of Schwann; the nuclei enlarge, become richer in chromatin, and penetrate between the fragments of the myelin. Simultaneously, the protoplasm increases in volume and fills up the empty spaces between the drops of myelin. The latter are surrounded by protoplasm and form around nucleated lumps. The nuclei of the sheath multiply by karyokinesis on the third and fourth days, and subsequently become very abundant in numbers. The decayed medullary sheath and axis-cylinders, which are in the form of small, round drops, are often mistaken for fat. A great part of these nuclei, surrounded by protoplasm, transform these cells into long spindle elements, the importance of which is variously interpreted. This feature will be considered when the process of regeneration is taken up.

According to Ballance and Stewart,[3] the various steps in the process of degeneration are as follows: (1) Cellular change; (2) changes in the axis-cylinder and medullary sheath.

Cellular Changes.—The first sixteen or eighteen hours after the division of the nerve there is a diapedesis of leucocytes in the entire distal segment, and also in a small portion of the proximal segment. In from three to fourteen days, connective tissue cells replace these leucocytes. Similar to the invasion of the nerve with the leucocytes on the second day after the injury, there is a marked proliferation of the connective tissue cells, invading the entire distal segment and a small portion of the proximal. On the fourth day the fatty substance of the myelin fragments is included within the substance of the connective tissue cells. The exact time in which the absorption is completed has not been determined, according to these authors. It is estimated, however, that the myelin disappears in about five weeks. After the complete absorption of the myelin, the connective tissue cells diminish and are outnumbered by the gradually increasing neurilemma cells. The remaining connective tisue cells become spindle-shaped and their destination is the formation of connective tissue fibers separating the chains of neurilemma cells. On the second day after the injury the neurilemma cells proliferate. At first their proliferating activity is greater than that of the connective tissue cells, and during this period they exert an absorptive action upon the degenerating medullary sheath. Later, their proliferating activity is surpassed by the connective tissue cells, the former continuing to proliferate in columns. The young neurilemma cells preserve the original longitudinal direction of their parent cells, and from their opposite poles they shoot out fine protoplasmic processes which gradually increase.

Changes in the Medullary Sheath.—Decay of the axis-cylinders begins on the fourth day, and is very pronounced on the seventh. Fine fibers have a greater resistance to disintegration than the coarse ones. The fragments of the axis-cylinders are absorbed by the connective tissue cells principally, and partially by the neurilemma cells. The degenerative process is not influenced by immediate suture, as it takes place uniformly. The muscle supplied by the divided nerve rapidly diminishes in weight.

Regeneration.—This begins, according to Ballance and Stewart, before degeneration is completed. At the end of the third week the peripheral segment contains a great number of elongated spindle cells, which shoot out axis-cylinders from their poles. The scar tissue contains cells whose ends interlace in an irregular manner, and form a new network of new axis-cylinders. The anastomosis of the axis-cylinder of the two ends of the nerve is accomplished at the end of four weeks.

SYMPTOMS.

These will depend on the location of the lesion, with reference to the involvement of other nerves or parts of the body. There is, however, a characteristic picture of a facial paralysis that can not be mistaken very well for any other condition, although we may have different degrees of paralysis, as a mild paresis or complete paralysis.

The characteristic picture (Fig. 4) shows the angle of the mouth drooped, the usual nasolabial fold obliterated; in fact, the whole cheek hangs flabby. The lower eyelid sags and the upper one lies closer to the eyeball; the eyebrow seems lowered as well as the skin covering the forehead—the usual creases of this region are wanting. Tears are found gathered in the corners of the eyes, and often seen trickling over the lower lid. The bulbar conjunctiva is frequently injected. So far as the patient is concerned, there is a most unpleasant appearance and sound when he begins to speak, to laugh or attempts to move the facial muscles. He can not close the eye, and when forced to do so, the eyeball rolls up, and one sees only the scleral portion. Asked to whistle or to blow up the cheeks, one finds that it is impossible for him to do so. The air will escape through the paralyzed side of the mouth. Patients complain a great deal of their food lodging within their cheeks, and find considerable difficulty in clearing the buccal surfaces. They generally use the other side of the mouth for mastication. Inasmuch as most of the cases are those where the nerve is injured above the chorda tympani, there are symptoms of perverted or loss of taste. A complaint frequently made is that of a metallic taste. The eye symptoms bother the patient a great deal, especially when exposed to the wind, and on first arising in the morning.

PROGNOSIS.

This depends absolutely upon the cause and location of the lesion. Suffice it to say, however, that in general the prognosis is bad and the course is a long and protracted one, except in that group of cases known as Bell's palsy, such as are classed as toxic neuritis, rheumatic, luetic or compressive. In the latter cases, when the condition is relieved, recovery takes place more readily, and even here one must give a very guarded prognosis. I have had a number of cases of the above type that resisted all sorts of local and general treatment, and only after three years did complete recovery take place. However, most of them recovered much sooner, between three weeks and six months.

Again, the prognosis depends on the treatment. I mean, specifically, surgical treatment, and when instituted. To be more specific, if one has a complete paralysis, with a positive reaction of degeneration, it is best not to wait too long before operating for fear of losing the muscular action, as the muscles tend rapidly to undergo trophic changes and waste away, after which an operation on the nerve will be practically useless. Another important point is the onset of paralysis, especially after operation. If it be gradual and take two or more days before development, the progress is better than if it has occurred as soon as patient comes out of the anesthetic, which condition is more grave.

REACTION OF DEGENERATION.

Normally, the facial, like other motor nerves, is affected by electrical stimulation as follows: The faradic current, when applied over the trunk of the facial, behind the ear or over the point of emergence of its branches on the face, causes a quick contraction of muscles supplied by these nerves. The electrodes may be applied directly over individual muscles with the same result. When a sufficient amount of galvanic current is applied over the nerve or muscles, contraction occurs every time contact is made, with the so-called "make" of the current; slightly stronger currents produce another contraction when the current is released or "broken." In all these stimulations the "cathode" or negative pole has been found to be the more active; that is, less current is required to get a contraction when the negative pole is placed upon the muscle to be stimulated; while the positive electrode may be put upon an indifferent point, preferably the sternum or back of the neck. The normal formula usually given is:

C. C. C. > A. C. C. = A. O. C. = C. O. C.

In which C. stands for cathode, A. for anode, C. for closing, C. for contraction, O. for opening.

C. C. C., cathode closing contraction.

> A. C. C., greater than anode closing contraction.

What do we mean by the reaction of degeneration, which is abbreviated by R. D.?

In this, the important points are that faradism fails to produce a contraction both through muscles and nerves, while galvanic currents produce no contraction of the muscles when applied over the nerve, but still cause muscular contraction when applied directly over the individual muscle. There is, however, a change in the character of the contraction. It has ceased to be prompt, lightning-like, but has become sluggish, peristaltic-like. A minor point is the inversion of the polar reactions. Whereas in the normal muscle the cathode is the more active in closing or making of the current, in the degenerated muscle the anode may be more active in closing and less than normal in the opening contraction, that is, the reverse of the normal muscle. This change, however, is unimportant. The time of occurrence of the reaction of degeneration is from eight to ten days following injury or disease of the facial nerve.

Immediately following injury, the nerve response may be even greater than normal; that is, less current is required to produce the effect. Authors have differentiated various degrees of reaction of degeneration according to the completeness or incompleteness of the reaction formula. The prognosis, therefore, is somewhat governed by the fact as to whether reaction of degeneration is complete or incomplete. The less complete the R.D., the better the prognosis.

TREATMENT.

The treatment must necessarily again depend on the etiology and pathology, as well as on the location of the lesion. In a general way, however, we can say the treatment is either expectant, local, general, medicinal, mechanical, or surgical. Taking up the cases systematically, as mentioned in the etiology, I will state that the local treatment will constitute in the first place, removing, if possible, whatever may be pressing on the nerve, as tumors, abscesses, necrotic bones, sequestra, and other foreign bodies. One of the frequent procedures necessary is a mastoid operation, and the removal of cholesteatomatous masses, sequestra and granulations over the region of the facial canal. Quite a number of cases of this sort will get well after such procedure.

Facial massage, vibratory massage, etc., may be systematically carried out, twice or three times daily for a half an hour at a sitting.

Electricity—a mild faradic current, is to be used as follows: The positive pole is applied over the sternum, and the negative used as the stroking electrode over the whole paralyzed area, once daily for three to five minutes, followed by galvanic current, the technic practically the same, using from five to ten milliamperes for three to five minutes.

The eye must be protected from external influences, as foreign bodies, dust and wind will irritate the condition; usually I give the patient a shield or a large lens (plain). Over night the eye should be tied up. A drop of argyrol, 5 per cent, and boracic acid irrigations are good adjuvants to prevent infection.

Thorough cleansing of the mouth and especially the inner surface of the cheek of the paralyzed side, as food particles remaining therein produce considerable irritation from decomposition.

The general treatment consists in tonics, particularly strychnin sulphate, 1/60 of a grain three times a day.

Eliminating and reabsorptive remedies should be used, as cathartics, sweat baths, potassium iodid solution.

A very wholesome nutritious diet, such as will leave the least residue, is to be advocated; rich condiments and red meats are to be restricted.

Strict attention to hygiene and exrcise is to be encouraged, particularly in the open air. A change of scene is to be recommended, that is, away from friends and acquaintances, because patients are very frequently sensitive about these conditions, and very often become morose, this condition causing delay in the recovery.

Of course, if it be a case of leutic facial paralysis, a thorough anti-syphilitic treatment by mercury and iodid of potash is to be instituted, associated with all the other general and local treatments required in that condition.

SURGICAL TREATMENT.

The indications for an operation on the facial nerve are confined to absolute paralysis, no matter from what cause and wherever the location of the lesion (lower segment), after all other methods have failed. The procedures are usually anastomosis with other neighboring motor nerves, as hypoglossal, spinal accessory and glossopharyngeal, in the order named.

The contraindications are: (1) When the general condition of the patient will not warrant such an extensive operation, and (2) when the other nerves, as hypoglossal, spinal accessory and glossopharyngeal, are not in good condition or accessible. (3) In cases of congenital absence of the facial muscles or nerve. (4) In long standing facial paralysis where one, in making the test of reaction of degeneration, can safely say that all the muscles supplied by the facial nerve are wasted and beyond the possible chance of regeneration. This last contraindication must, however, be modified in as much as there are cases on record (Hackenbruch) in which the above conditions existed, which were operated upon and recovered.

The historical data of neuroplasty may be briefly stated as follows: Probably Ballance and Stewart were the first to have united the facial and spinal accessory nerves for the cure of facial paralysis, in 1895, but did not publish it until 1903.[4] In 1898, at the suggestion of Fouret, Foure[5] united the facial with the trapezius branch of the spinal accessory. Experimentally, P. Manasse,[6] in 1900, and about the same time Barrago Ciarello[7] accomplished satisfactory results on animals in uniting the facial with the spinal accessory. Robert Kennedy,[7] in 1900, operated on a man by uniting the facial with the spinal accessory. Harvey Cushing[8] performed the same operation in 1902. After that, quite a number of similar operations followed. The hypoglossal, however, was not used until about 1902-3, as cases reported by Ballance and Stewart (*British Med. Journal*, May, 1903). Koerte,[9] Nicoll,[10] Frazer,[11] Bardenhauer,[12] Schaefer,[13] of Edinburgh, suggested the union of facial and glossopharyngeal.

Herewith follow the cases tabulated by Murphy, with some additions by myself from literature and personal communication as well as cases operated on by myself:

NERVE ANASTOMOSIS FOR THE CURE OF FACIAL PARALYSIS, TIC, OR SPASM.

A.—Experimental.

CASE 1.—Paul Manasse. (Munk's Laboratory.) (*Arch. f. klin. Chirurgie*, vol. lxii, p. 822.) Seven dogs operated on, but only five considered in final results. Spinal accessory freed from its implantation into sternomastoid muscle to atlas; end-to-end anastomosis of central spinal accessory with peripheral facial divided at the stylomastoid foramen. Four silk sutures passed through quarter of the thickness of spinal accessory.

Results.—Clinical results not sufficiently satisfactory. In first three months there was no faradic response; the latter obtainable after four months. Electric stimulation of central spinal accessory was followed by response in peripheral facial. Histologically, new axones penetrated the facial from the central spinal accessory. Manasse thinks that "months" is an insufficient time for voluntary motion and disappearance of atrophy. "Years" would lead to perfect restoration.

CASE 2.—Barrago Ciarello. (*Policlinico,* 1901, No. 3.) In two dogs. (a) Anastomosis between central end of spinal accessory and peripheral of facial. (b) Anastomosis between the central end of vagus and peripheral end of facial.

Results.—(a) Restoration of function commenced after four months, and was almost completely restored in six. There was still some muscular atrophy left. Reaction of degeneration disappeared. (b) Same results. Ciarello believes that spino-facial and vagofacial anastomosis is promising in man in recent facial paralysis.

B.—Clinical.

CASE 1.—Charles A. Ballance, Hamilton Ballance, and Purves Stewart. (*Brit. Med. Jour.,* May 2, 1903.) Facial paralysis. Following operation for suppurating mastoiditis. Spinofacial anastomosis. (Operation by C. A. Ballance.)

Results.—After several years, muscles responded to galvanic and faradic currents. Still some drooping of angle of mouth and widening of palpebral aperture. Movements always associated; voluntary independent movements of muscles of face not possible.

CASE 2.—Ballance and Stewart. Facial paralysis. Otorrhea of twenty years' standing. Spinofacial anastomosis performed. (Operation by C. A. Ballance.)

Result.—Some improvement after ten months.

CASE 3.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis performed (after six months). Anastomosis of central end of spinal accessory with side of facial. (Operation by H. Ballance.)

Results.—All muscles of face responded to faradic currents after four weeks. Three months later, some voluntary motion. Three sutures of French silk used.

CASE 4.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis, four months after the initial paralysis. (Operation by C. A. Ballance.)

Results.—Faradic response after nine months. Associated movements only. After twenty-one months, voluntary independent elevation of inner end of eyebrow.

Case 5.—Ballance and Stewart. Facial paralysis. Fracture of skull, due to a fall of fifteen feet. After thirty-five months. Spinofacial anastomosis. (Operation by C. A. Ballance.)

Results.—Slight voluntary movements in muscles of face if shoulder was elevated under resistance.

Case 6.—Ballance and Stewart. Facial paralysis. Following operation for otitis media. Spinofacial anastomosis six months after initial paralysis. (Operation by C. A. Ballance.)

Results.—No improvement; no electric response; no return of independent voluntary or associated movements.

Case 7.—Ballance and Stewart. Facial paralysis. Following the removal of a cystic tumor of lower jaw. Operation twelve months after initial paralysis. (By C. A. Ballance.) Anastomosis of peripheral end of facial with side of hypoglossal.

Results.—No improvement at the time of operation. Atrophy of half of the tongue.

Case 8.—Faure. January 28, 1898. (a) *Gazette des Hopitaux,* 71eme annee, 1898, p. 259. (b) *Revue de Chir.,* 1898, t. xviii, p. 1098. Facial paralysis. Injury. Eighteen months later, anastomosis of facial with accessories.

Results.—Slight improvement some time after the operation. "Half successful."

Case 9.—Robert Kennedy. Phil. Trans. of the Royal Society, series B, vol. cxciv, p. 127. Facial tic (facial hemispasm). Spinofacial anastomosis. End to side.

Results.—Cured. After 470 days, patient able to close eye. Muscles regained volume, and contracted voluntarily.

Case 10.—Hackenbruch (Wiesbaden). *Verhandl. der deutschen Gesellschaft für Chir.,* 1903, bd. xxxii, p. 231. Facial paralysis. Sudden paralysis of facial, three months' standing. Seven and three-fourths years after appearance of paralysis, Spinofacial anastomosis.

Results.—Improvement after four and one-half months. After nine months, patient could draw the angle of the mouth downward.

Case 11.—Körte. (*Deutsche med. Woch.,* April 23, 1903.) Facial paralysis. Following operation for ostitis of mastoid process. Hypoglosso-facial anastomosis.

Results.—Good after one and one-half years, but associated movements present.

Case 12.—W. W. Keen. Phila. Neurol. Soc., Feb. 24, 1903. (*Jour. of Nerv. and Ment. Dis.*, Feb. 24, 1903, p. 366.) Facial paralysis. Struck by a glass bottle. Five weeks after accident. Spinofacial anastomosis.

Results.—"Perfectly smooth recovery."

Case 13.—Harvey Cushing. (*Ann. of Surg.*, May, 1903.) Facial paralysis. Revolver-shot behind ear, involving mastoid. Operation forty-three days later. End-to-end spinofacial anastomosis.

Results.—Complete restoration of function.

Case 14.—Hackenbruch. (*Arch. f. klin. Chir.*, 1903, Bd. lxxi, p. 631.) Facial paralysis of seven years' and nine months' standing. Spinofacial anastomosis by implanting two-thirds of spinal accessory into facial.

Results.—Electrical response in paralyzed muscles. Voluntary contraction independent of those of trapezius.

Case 15.—James H. Nicoll. (*Lancet*, 1903, p. 956.) Convulsive tic. Anastomosis of facial with hypoglossal.

Results.—No improvement at the time of communication.

Case 16.—Szeyner. (Abstracted in *Fortschritt der Mediz.*, 1903, p. 1060.) Facial paralysis. Shot; attempt at suicide. Facial anastomosis with a flap taken from spinal accessory.

Results.—Voluntary contractions after three months; able to close eye, and whistle.

Case 17.—Charles H. Frazier. (*Univ. of Pa. Med. Bull.*, 1903-4, vol. xvi, p. 306.) Facial paralysis. Gunshot wound. End-to-side anastomosis between peripheral facial and side of hypoglossal.

Results.—Too recent to report conclusively on the case.

Case 18.—Glück. (*Berl. Gesell. f. Psychiatrie*, May 11, 1903.) Facial paralysis. Division of facial during mastoid operation for otitis media. End-to-side anastomosis between peripheral facial and side of spinal accessory.

Results.—Regeneration of facial. Associated movements.

Case 19.—A. I. Bouffleur, Chicago. Personal communication. Facial paralysis due to gunshot wound (attempt at suicide), June 14, 1904. The tip of mastoid was cut off by the bullet. Division of lower branches as well as of common trunk of facial. End-to-end anastomosis between spinal accessory and facial, performed four weeks after injury.

Result.—Case lost sight of.

Case 20.—Dr. O. Facial paralysis. Following operation for otitis media. March 10, 1904. End-to-end spinofacial anastomosis.

Results.—Muscular atrophy not improved. Patient has associated movements only in muscles of face.

Same case as above. J. B. Murphy. Facial paralysis. Following operation for otitis media. March 10, 1906. Freed nerve from scar tissue and imbedded it in muscle.

Results.—Muscular atrophy improved, and voluntary motion of face is manifest. The associated movements continue. Patient was reoperated on by the author.[1]

Case 21.—Löhlein. (Communication, *Vereinigung der Chirurgen,* Berlin, July, 1904.) Facial paralysis. Otitis media. Anastomosis of facial with spinal accessory.

Results.—"Excellent results." No associated movements.

Case 22.—Bardenheuer. (*Münch. med. Wochenschr.*, 1904, No. 28.) Facial paralysis of ten years' standing. End-to-end anastomosis between peripheral facial and side of hypoglossal.

Results.—"Very good." Tongue paralyzed. No associated movements.

Case 23.—Bardenheuer and Lambeth. (*Festschrift zur Eröffnung der Akademie für praktische Medizin zu Köln*, 1904, p. 219. Facial paralysis. End-to-end anastomosis between peripheral facial and side of hypoglossal.

Results.—Good.

Case 24.—Mintz. (*Zentralbl. f. Chir.*, 1904, No. 22, p. 684). Facial paralysis. Trauma during mastoid operation for otitis media. Anastomosis of facial with spinal accessory.

Results.—Complete restoration of motor function. Good closure of eye.

Case 25.—Villar. (*Gaz. hebd. des. Sc. Méd.*, Bordeaux, Feb. 7, 1904.) Facial paralysis. Trauma to facial during mastoid operation for otitis media. Anastomosis of facial with spinal accessory.

Results.—"The results were astonishing, as patient was able to move his lid on next day after operation. This rapid improvement, however, did not remain permanent."

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[1] The point of anastomosis found surrounded by great mass of connective tissue.

Electrical irritation of central end of spinal accessory produced contractions in muscles of the face, showing that new fibers had grown from the spinal accessory into facial, but that impulses could not travel, on account of surrounding connective tissue. Point of anastomosis was freed and protected by soft tissues. Good results.

CASE 26.—Alexander. (*Wien. med. Wochenschrift,* No. 11, 1904.) Facial paralysis. Otitis media paralysis of five years' standing. End-to-side anastomosis between divided facial and side of hypoglossal.

Results.—Paralysis of half tongue. No improvement at time of communication.

CASE 27.—Alfred S. Taylor and L. Pierce Clark. (*Jour. Am. Med. Assoc.,* March 24, 1906.) Facial paralysis. Suppurative mastoiditis. The peripheral segment of facial inserted into a slit of hypoglossal.

Results.—Improvement after twenty-five months. The anastomosis was protected by Cargile membrane.

CASE 28.—Same. Facial paralysis. Paralysis followed a severe earache. Same operation.

Result.—Improvement.

CASE 29.—Same. Facial paralysis. Suppurative mastoiditis. Same operation.

Result.—Improvement.

CASE 30.—Same. Facial paralysis. Bell's palsy. (Paralysis twelve years' standing.) Same operation.

Results.—Improvement. Although paralysis existed for twelve years, facial nerve was normal in size and aspect.

CASE 31.—Same. Facial paralysis. Suppurative mastoiditis. Same operation.

Result.—No improvement.

CASE 32.—A. I. Bouffleur, Chicago. Personal communication. Facial paralysis resulting from fracture of mastoid, concomitant with fracture of skull (1903). Nerve-trunk intact, as far as continuity is concerned. End-to-end anastomosis between spinal accessory and facial. Performed September 11, 1906.

Results.—Some improvement shortly after operation, inasmuch as fluids did not run out of the mouth from paralyzed side. Time too early to give definite results.

CASE 33.—J. B. Murphy. Present paper. Facial paralysis. Division of facial during mastoid operation for otitis media. End-to-end anastomosis between peripheral facial and central spinal accessory.

Results.—Evidence of returning motion pronounced. See history. Too recent for complete recovery.

Spiller, University of Penn., *Med. Record,* November, 1903.

Rothmann, *Berliner klin. Woch.,* 1903, No. 34.

Bernhardt, *Berliner klin. Woch.*, 1903, No. 34.

Koster, *Berliner klin. Woch.*, 1903, No. 34.

Gradenigo, *Laryngoscope*, 1907.

McArthur, Eisendrath, Ochsner, Chicago surgeons, have done this operation, but for some reason have not reported their results.

Beck, J. C., five cases of facial hypoglossal anastomosis. (Present paper.)

GENERAL POINTS AS TO NERVE ANASTOMOSIS.

In the first place, a clean, aseptic field for primary union of the sutured ends is practically necessary to obtain rapid regeneration through the anastomosed ends; proper protection (as shown in Murphy's cases) of the anastomosed ends, to prevent access of cicatricial formation about them, by placing into a neighboring muscle; absolute rest to the parts, almost immobilization of the head for the first few days after operation, to prevent severance or traction on the united ends.

The methods of anastomosis most frequently employed are:

1. End to end.
2. End to side.

The most preferable mode is that of implanting the exposed stump on the paralyzed side into a niche of the living nerve; this niche should have its axones about 1/2 to 2/3 cut transversely, in order to obtain direct contact and union, and, secondly, so that absolute loss of function does not result in the muscles supplied by the live nerve, as, for instance, the tongue would only be partially paralyzed when the hypoglossal is used.

SUTURE OF NERVES.

Either primary or secondary. By "primary suture" is understood suture of the two divided ends immediately after the accident or within the first twenty-four hours. Secondary suture is the approximation of the two divided ends within several months or years after the injury. The older surgeons base their practice on the theory that a peripheral segment which has been disconnected from the central for a long period of time is permanently degenerated, and an attempt at union would be futile. The most modern surgeons, better informed on the histology and physiology of the peripheral nerves, and especially those who believe in peripheral regeneration, are of the opinion that new nerve fibers regenerate in the peripheral segment, even if it has remained disconnected for a considerable period of time. The fibers are imperfect, and as soon as they are united with the central end, under the influence of central impulses they develop to anatomic perfection and to normal physiologic function. The limitations of time in which this restoration may take place has not been definitely determined, but restoration of functon has been secured many years after division.

In any operation upon the nerve trunks, one must, first of all, identify the nerve; otherwise the operator is in the dark, and the final result will undoubtedly be compromised. This identification can easily be accomplished by making use of the nerve excitor. This is composed of a metallic cylinder which can be attached at one end with the poles of a faradic battery, while the other end is provided with two platinum needles or wires, three centimeters in length, and situated a distance of half a centimeter from each other. These tips can be easily sterilized, and should always be at hand when operating upon the nerve trunks. The importance and the value of this instrument can be appreciated when we think of the conditions of the tissues in an old and extensive injury in which they are all matted and bound together by connective tissue. The structures have lost their natural color, and the anatomic relations and identification of nerve trunks becomes an impossibility without the use of the excitor.

Operative Procedure.—The first essential is the removal of all connective tissue that caps the nerve endings to a degree sufficient to expose clearly the fasciculi and neurones, even though this shortens the nerve beyond the possibility of direct end to end suture. The axis-cylinders or nerve fibers will span a considerable distance under favorable conditions, but they will not penetrate connective tissue masses, so that it is absolutely necessary to have all the cicatricial or new-formed connective tissue removed from the nerve ends. The failure of this removal has contributed more than any other element to the failure of restoration of function after nerve suture. This, of course, has reference to secondary suture.

The needle used for suturing should be a round intestinal needle, without cutting edges. Suture material should be fine linen, silk, kangaroo tendon, or slowly absorbable catgut. The suture should not be passed through the substance of the nerve, but through the perineurium, and also through the surrounding connective tissue. This point is very essential for success. The trauma produced by passing the needle through the substance of the nerve, and especially if a slight infection takes place, causes a proliferation of connectice tissue which compromises the restoration of function. Figure 5 shows the various methods of suture.

If the nerves face each other, and are not separated by a considerable gap, a slight traction established when the muscles are completely relaxed, will make it possible easily to approximate the ends. A little stretching of the ends can be easily made without any prejudice to the ultimate result. If the distance is considerable between the ewo ends, the nerves can be united in several ways. Létievant is the originator of "suture a lambeaux," or flap-suture. Foreign bodies, nerve segments from other animals, portions of segments of spinal cord from small animals, have been interposed between distant segments. In the last ten years, in cases of considerable distance between ends, the divided peripheral end was implanted into a neighboring healthy nerve, which was only partially divided. In several instances, especially where the trauma has compromised the nerve and bone, in order to be able to approximate the two ends a segment of bone was resected, that the divided ends might be brought in contact (Keen, Trendelenburg). Büngner interposed a piece of human brachial artery between the segments of the peripheral nerve in a dog. After 43 days the space between the two divided ends of the sciatic was filled with new nerve fibers. Bungner ascribed to the artery the role of a director and medium along which the nerve fibers can travel in a straight direction. Nothaft interposed a segment of rabbit's aorta between the two ends of the sciatic, from which he had excised one-half centimeter. When the gap between two ends is considerable, the contact can be established by foreign material. In a case of injury to the radial, Glück, in 1888, interposed catgut bundles between the ends, which were separated by a gap of 5 cm. The gap was filled in six months, or, as he says, there was a neurotization of the catgut bundles. The result was good.

On the strength of Forsman's observation on "positive neurotropism," several surgeons and experimenters have inserted the two ends into a tube of decalcified bone, previously sterilized; this has enabled a more rapid union of nerve fibers.

"Suture a Distance."—This procedure originated with Assaky (Roumania). By this is meant the interposition of catgut fibers between the two ends of a divided nerve where a considerable gap exists. In Assaky's experiments (4 dogs, 2 rabbits), in the dogs favorable results were obtained in from 67 days to 4 months. The greatest segment successfully excised was 3 cm. Once he resected 6 cm. and interposed catgut fibers between the ends; no regeneration occurred after 39 days. If the distance is considerable between the segments, a piece of muscle may be interposed, or the two segments may be imbedded in a muscle from the vicinity in such a manner as to place the cut surface of each end face to face. This has been done by Levings, of Milwaukee, and several times by Murphy. The ideal results in nerve suture are obtained, as in the repair of all other tissues, by an exact juxtaposition of similar histologic elements, end to end. This requires the least interposition of new-formed connective tissue, and in nerve union it is of special importance.

Protection for United Nerves.—The ends of the nerves which are either in perfect apposition or face each other from a distance may be protected from the invasion of connective tissue from the side by sterilized tubes. Decalcified bones were used by Vanlair. Payr used tubes of magnesium. Lotheisen prefers to protect the nerves with tubes of gelatin previously hardened in a two per cent solution of formalin. The gelatin tubes, after being treated by this solution, are preserved in 96 per cent alcohol; the lumen should be from 4 to 9 mm. Gelatin tubes can be sterilized for two hours by dry heat of from 110° to 120°. It has been my practice to protect the nerves from invasion of connective tissue by covering the point of suture with fascia, muscle substance, or fatty tissue. In the wrist or ankle areas, where muscle and fat are not available, gelatin and decalcified bone are good materials. The blood vessels of animals, always at hand, are good for this purpose. A mass made of equal parts of oil of sesame and paraffin, with a melting point of 107° is especially good. This can be flattened out with a spatula almost as thin as paper and wrapped around the point of union, completely sealing the ends so that the neuroglia can supply the only immediate agglutinative material, which is readily penetrated by the axones.

Foramitti protected the points of anastomosis by surrounding them with arteries of the calf. The union is always perfect, and the point of anastomosis entirely free from the connective tissue in the vicinity. There is an insignificant adhesion between the intima of the artery and the sheath of the nerve. The arteries are prepared for this purpose in the following manner: A segment of artery from a calf is placed on a glass rod and hardened in 5 to 10 per cent of formalin solution for 48 hours, then it is washed in water for 24 hours, afterwards boiled for 20 minutes in water, and finally preserved in 95 per cent alcohol.

An ever-available material for this purpose is the membrane lining the inner surface of an egg-shell. The Cargile membrane may be used for the same purpose, as may also the peritoneum of a rabbit. Recently Murphy conceived the idea of wrapping around the point of anastomosis a roll of paraffin tape or a mixture of paraffin and wax, or simply a wax tape; however, these foreign materials were never used when a muscle or fascia could be obtained, either by flapping or splitting the muscle and inserting it where a normal muscle belly is directly available. The use of inorganic material is always somewhat hazardous.

THE SURGICAL ANATOMY OF THE FIELD OF OPERATION.

(Fig. 6.)

1. *Facial-Hypoglossal Operation.*—After the skin and superficial fascia and platysma are retracted, a number of small veins are encountered, branches of the anterior and posterior facial, external and internal jugular. The anterior border of the mastoid process is located, and the sterno-mastoid muscle. Then the parotid gland is exposed with the ramus and angle of the lower jaw. On separating the sterno-cleido-mastoid muscle backward, one comes to the posterior belly of the digastric, which, when drawn under the lower jaw, will expose the submaxillary gland. Now the common sheath of the internal jugular vein is seen with the internal and external carotid arteries below and anterior to it. On slight retraction backwards of the sterno-cleido-mastoid muscle and internal jugular, one can see shining through a loose areolar tissue, a whitish band, about one-eighth of an inch in diameter, which, when laid bare, is the hypoglossal nerve. This can be followed under the posterior belly of the digastric. At this point it lies on the internal carotid and some of the branches of the external carotid.

as it comes down and curves forward, particularly the occipital artery.

The parotid region will show on its posterior border, the facial nerve entering and passing transversely under the auditory meatus to the anterior border, and under surface of the mastoid process. It lies at this point on top of the stylo-hyoid muscle, consequently the styloid process, which can always be felt. The transverse process of the atlas is situated at this point, and is the landmark or location of the beginning spinal accessory nerve.

2. *Facial-Spinal Accessory Operation.*—At first, the same structures come to view, but in order to obtain the view of the spinal accessory the internal jugular is drawn forward or towards the larynx, and the sterno-mastoid muscle is raised, when the upper branch, or the one supplying the latter muscle, is seen, and the greater branch passes down to the trapezius. (Shoulder.)

TECHNIC OF THE FACIAL-HYPOGLOSSAL ANASTOMOSIS.

After the usual local as well as general preparation the patient is placed under a general anesthetic.

Instruments.—Two knives, one regular size and one very small; one dozen fine artery forceps; two pairs of tissue forceps; two pairs of anatomic forceps; one Kocher director; one simple grooved director; two small dull retractors; two larger dull retractors; one aneurysm hook; two pairs scissors, one straight, one curved; one pair of fine-pointed conjunctival forceps; one special facial nerve dissecting instrument (Fig. 7); one-half dozen fine small needles (round); one needle-holder.

Material.—Sutures, finest of silk, catgut, silkworm gut, decalcified chicken bone tubes, Cargile membrane, paraffin tape.

An incision is made, beginning close back of the auricle, midway between its upper and lower margins, carried downwards and slightly forwards in the direction of the hyoid bone. The length of the incision should be about four inches, and carried at first through the skin, superficial fascia and platysma muscle. These cut margins should be dissected loose and retracted. The external jugular vein, with its anterior and posterior facial veins, is exposed, ligated above and below, and severed. The deep fascia is carefully incised, preserving any of the branches of the superficialis coli nerves; the anterior border of the sterno-cleido muscle is exposed and followed upwards to the tip of the mastoid process. The posterior belly of the digastric is next exposed and the posterior border of the parotid gland by forcibly retracting the auricle and the skin over the ramus of the lower jaw forward. The anterior border of the mastoid process is followed along towards the tip into the depth, so as to reach the deeper layer of the parotid gland and to look for the entrance of the facial nerve into the latter, about on the level of the lower border of the external auditory meatus. The greatest mistake is made in looking for the facial nerve too near the surface and too far down from the ear. (One often finds here a superficial cervical branch.) After finding the facial nerve, which is recognized as a whitish cord, about the size of a Eustachian catheter No. 2, it is hooked up by means of the aneurysmal hook, and followed backward and upwards towards the stylo-mastoid foramen. This can best be located by following back the posterior belly of the digastric and the stylo-hyoid muscle immediately under it. The highest point of these two muscles will locate the foramen and the exit of the facial nerve. (Fig. 8.) In order to follow the nerve as high up into the foramen as possible, I have adopted and modified the ring instrument as devised by Mayo for dissecting varicose veins subcutaneously, and have added to it its ability to grasp. It is used in the following manner: Slip the ring over the nerve and follow it down to the foramen as high as possible and pull it out of canal. Place a sponge soaked in warm salt solution about it and allow it to remain in the field. Next the hypoglossal is located by retracting the posterior belly of the digastric upwards under the lower jaw and the sterno-cleidomastoid backwards. The common sheath of the vessels is exposed. This, with the sterno-cleido-mastoid muscle, is bluntly retracted. By bluntly dissecting some of the loose areolar tissue over the internal and external carotids, which can be felt pulsating with the finger, one can see a whitish structure passing over the external carotid just where the occipital artery is given off. This is the hypoglossal nerve which can be traced at this turn under the internal jugular and internal carotid upwards for a short distance, where it is lost sight of in its direction towards its foramen vertically into the cranial cavity. The distal course of the hypoglossal is forwards towards the tongue. At this point the technic will differ, whether we do an end to end or end to side anastomosis.

End to End.—(Fig. 9.) Follow the hypoglossal in its forward course to the tongue. The same ring-formed scissors may be used as for the facial. The nerve should be severed at the point, so as easily to meet the facial stump. The next step is to bring the cut ends into perfect apposition and suture them. One can use any of the methods described in the suture of nerves. The anastomosed nerves are now carefully placed into a pocket made within one of the muscles, preferably the posterior belly of the digastric, and a few superficial catgut sutures inserted, closing this pocket. All the structures of the neck are carefully replaced in their anatomic positions and wound closed without drainage, unless pus has been encountered, as in a suppurating gland, etc.

End to Side.—(Fig. 10.) Bring down the amputated facial nerve stump and find the location on the hypoglossal where the anastomosis will be best, that is, without producing any tension on either the facial or hypoglossal nerves. Having found that point, an incision, one-quarter inch long, is made with the small knife into the nerve sheath parallel with the course of the nerve. With the same knife about one-half of the axis-cylinders are several within the sheath, that is, transversely, in order that when the facial stump is later implanted there will be direct axone contact, and thereby proper union, and still some of the fibers be left intact to perform part of the hypoglossal function. The next procedure is the preparation of the facial stump. The nerve sheath should be slit for about one-eighth of an inch and retracted so as to expose the stump, that is, its axis-cylinders. The sutures are placed within it, but only through the sheath. Any method may be selected for suture of the nerves utilizing the slit. Fine conjunctival forceps are used by the assistant to separate this slit by allowing them to spring open. After the sutures are tied, these anastomosed nerves (fig. 11) are placed within a pocket in a muscle (posterior belly of digastric) and treated in the manner already indicated. The wound is closed in the same way as in the other procedures.

TECHNIC OF THE FACIAL-SPINAL ACCESSORY ANASTOMOSIS.

The first steps of the preparation and operation are the same, except that the skin incision is not made so far curved forward, because the spinal accessory nerve lies more posterior than the hypoglossal. Locate the transverse process of the atlas; by well retracting the sterno-cleido-mastoid at the mastoid tip, one can hook up the spinal accessory nerve. If it is decided to use the trapezius branch, then that is dissected and severed, brought upwards and united to the facial. The anastomosed nerves are then placed within a pocket formed within the sterno-cleido-mastoid, or the posterior belly of the digastric muscle, and secured with two catgut sutures to prevent cicatricial formation at the new-formed union of the nerves, thereby preventing bad results.

POST-OPERATIVE OBSERVATIONS AND TREATMENT.

There are considerable soreness and pain in the regions operated on, and patients complain of some difficulty in swallowing and speaking for the first day or two. This is especially so if the hypoglossal is chosen. If closely questioned, most of the patients will state positively that they feel a sort of a tone in the paralyzed side of the face, but this soon disappears, and does not return until later, when gradual regeneration has taken place. Patients should be kept absolutely at rest for about a week, and as little motion of the head as possible should be made in order to insure good and rapid union of the nerve ends. The diet may be regular after the first forty-eight hours, if the patient is able to swallow well. The stitches may be removed at the end of a week or ten days.

Electric treatment should be begun as soon as the patient is walking about, in order to keep up the strength of the muscles and to stimulate the nerves. Mild faradic and galvanic currents are used as before operation. In case the hypoglossal is used, about the first signs that one observes of return of function are a sort of contraction of the angle of the mouth, especially during the act of swallowing, and this may be observed as early as three months after operation. Gradually, the upper lip, and, finally, the eyelids take on a toned-up and straight condition, contracting when the patient swallows. The patient declares very early after the operation that the food does not lodge within the cheek, and mastication on that side is so much more satisfactory. All other symptoms, such as lacrimation, etc., disappear. The test to reaction to degeneration shows return to the normal, that is, as described before. In case many nerve fibers of the hypoglossal have been cut, or all of them, there will be a tongue paralysis, which, outside of some speech difficulty, causes no untoward symptoms.

In case the spinal accessory nerve is used, then there will be the movements of the face associated with the movements of the shoulders, unless the whole trunk is used; then, of course, there will be a shoulder droop, a condition not very desirable, and perhaps worse than the facial paralysis. Otherwise, the same course of treatment is carried out as in the hypoglossal, and the same observations are made in reference to associated function.

REASONS FOR PREFERENCES AS TO NERVES SELECTED, AND METHODS.

An end to side anastomosis is preferable to any other method because the function of the tongue is not entirely destroyed and the desired cosmetic result on the face is obtained. The cortical, as well as nuclear, centers of the facial and hypoglossal nerves are much closer to one another, and it is believed that the function of the one is more easily transferred to the other than is the case with the spinal accessory. It is believed that the associated movements between the face and tongue on swallowing are more likely to be again disassociated. Again, the act of swallowing can better be contracted than the movement of the arm; it is very embarrassing to the patient to have his face move every time the arm is moved.

It is claimed that the end to end method is preferred because more axones come in direct contact with the opposite nerve, more rapidly unite and regenerate. The spinal branch is used in preference to the hypoglossal because it is easily located and on account of its close proximity to the exit of the facial.

As to the use of the glossopharyngeal for anastomosis with the facial, I have been able to find only one reference.

REPORT OF CASES.

Case 1.—Miss H., 42 years old. Has had a suppuration of her right middle ear since childhood, when she probably had Bezold's mastoiditis, as evidenced by a scar on her neck from multiple incisions. She came to me first in June, 1905, complaining very bitterly of pains in her ear and right sided head pains, foul smelling discharge, and a crooked face which she said had been existing only for a few months. She had been coughing for some time and generally feeling bad, losing flesh, and having night sweats. One of the principal complaints was a dizziness and a tendency to fall at times toward the right side. There was nothing important that could be elicited from the family history.

Examination.—A very frail, ill-nourished woman, weighing about 75 pounds. Temperature 99 1-2° (P. M.); pulse fair quality, and about 100. Nasal and throat findings negative. Right ear—external canal, negative, containing a fair amount of thick pus of a fetid character. After thoroughly removing it a practically normal tympanic membrane was observed, except in the lower posterior margin, where there was a small fistulous opening filled with granulations. Externally in the occipital regions marked tenderness was elicited by deep percussion on pressure. Test of hearing showed practically normal hearing through the whole range of Bezold, Edelmann, a positive Rinne. The left ear was also discharging some, but the patient knew nothing about it. There was a small perforation in the lower anterior quadrant. The hearing here was somewhat reduced and a slight negative Rinne-Weber lateralized to the left. After observation and local treatment for a short time, I decided to operate inasmuch as the patient complained more of her pains; especially when she attempted to lie on the right side of her head, she could not stand the pressure over the occipital region.

Operation.—Under general anesthesia the usual steps in radical operation were followed. When the annulus was resected and the middle ear cavity was explored, one could detect a necrotic area in the lower posterior portion of the middle ear, and that portion corresponding to the anterior extremity of the floor of the antrum, or posterior semicircular canal and the facial ridge. This area was very carefully curetted of the granulations, but I could not determine whether the semicircular canal or the facial nerve was injured at this point. I then proceeded to expose the painful area over the occipital region, and therefore made a tongue-shaped flap, with the base at the neck, or say an inverted U incision. Peeling this down, including the periosteum, we had a fearful venous hemorrhage from the region of the lateral sinus. This was controlled by compression, and on closer examination of the flap, we found a flake of bone, the outer table of the lateral sinus. This bone, however, appeared healthy. I then exposed the sinus farther back toward the torcular herophili and by compression controlled the bleeding point. On closer observation I found some granulations about this lateral sinus and a fistula.

By passing a flexible blunt pointed probe into the fistula it came out in the middle ear at the point spoken of above, namely, the lower posterior portion. Leaving this probe *in situ* I chiseled down upon it in order to transform it into an open wound. I firmly believe that the facial nerve was destroyed by the diseased process of the bone, or in any event I would very likely have injured it beyond the chance of spontaneous recovery, in order to eradicate the diseased process. The usual steps were then taken in finishing the plastic, having previously relieved the compression from the lateral sinus and tamponed the bleeding point of it. A retroauricular opening was left, in order to remove that tampon. The patient made an uneventful recovery and was perfectly free from pains and dizziness, but retained her facial paralysis. The reaction of degeneration showed plus, that is to say, no reaction resulted from the faradic current, when either applied to the nerve trunks, or muscles of the face, but there was a slow contraction of the muscles when the galvanic current was put on. That showed that the muscles were there and if union between the facial and another motor nerve took place, we could hope for results.

The photograph fig. 12 shows the condition and appearance before operation.

In the spring of 1906 the operation for facial-hypoglossal anastomosis was performed. Dr. Goldstein, St. Louis, Dr. Fleming, Los Angeles, and Dr. Owens, San Diego, California, were present and assisted. Reported in *Laryngoscope,* 1907. The operation as described in the previous chapter was performed and patient made an uneventful recovery.

Observation.—There was some soreness in swallowing and speaking which disappeared after two days. Patient declared voluntarily that there was a feeling in the right side of her face more like the other side than before operation; but this disappeared in a few days. I am inclined to believe now that that was from the reaction of inflammation in the tissues about the neck. No visible change could be noticed for about ten weeks, when a sort of a contraction of the right side of the face was noted and when patient was asked to swallow, this contraction was more apparent. From this time on, this phenomenon became more clear and gradually this tonicity was noticed over a greater portion of the face until about five months after operation; the eyelids, upper and lower, took on the tonic condition. At this time whenever the patient only thought of swallowing, the face would move and I began to think that that would be very detrimental to the success of this operation, but the patient soon learned to disassociate these movements and at the end of the year she could swallow by very slightly moving the face. At this time the test of reaction to degeneration was practically that as is found in normal cases. There was, however, this fact shown, that whenever patient laughed or smiled the healthy side of her face would respond, whereas the paralyzed side would lag and patient would have the characteristic appearance of facial paralysis. (Fig. 13.) There never was a voluntary movement of the facial muscles without the act of swallowing. The photograph, Fig. 14, which was taken about a year after operation, shows very well the patient as recovered so far as the cosmetic purposes go.

About this time the patient's cough again became generally worse and she decided to go to the country to be out in the fresh air. She wrote to me several times that she had lost her cough and was getting along well. On December, 1907, I wrote to her to come to Chicago as I wished to present her in connection with another case, and she came. When I examined her at this time, I found her in good health and her face straight. Two days later I was notified that she was taken to the hospital with acute lobar pneumonia, under the care of Dr. J. B. Herrick.

On the 10th day of the disease the patient died. The following day I made the post-mortem examination with these findings: General emaciation of the body. The face appeared symmetrical; field of operation of ear and anastomosis showed nothing different from antemortem. Calvarium section and removal of brain. No gross pathologic changes of the brain, venous sinuses, meninges, or bony structures in the vicinity of the temporal bones within the cranial fossae. Neck dissection of nerve anastomosis. Tracing the anastomotic union of facial hypoglossal into the substance of the posterior belly of the digastric and liberating it, I found perfectly firm union of these two nerves. Examination of the right thoracic cavity showed the whole lung solid and old scars and some recent tubercular foci which were excised for histologic examination. There was some turbid fluid in the pleural cavity. Pleuritic adhesion in the postapical region. Left thoracic cavity showed some recent consolidation near the base, and an old healed tubercular apex. Heart apparently normal. Intraabdominal viscera negative.

Histologic examination of lung structure showed characteristic changes of acute lobar pneumonia, and characteristic tubercular lung disease of the apex regions.

I then excised *lege artis* the wedge of the temporal bone including the structures of the neck which demonstrated the union of the facial-hypoglossal anastomosis, and herewith present it, after carefully dissecting and preparing it, with the following findings:

The cavity of the middle ear, attic, aditus ad antrum and antrum are perfectly healed and epidermized. The posterior semicircular canal is plainly seen where it was exposed and opened, and now is healed. The region of the ostium tubae tympanicae is firmly closed off from the pharyngeal region. The resected (mastoid process) fistula from the region of the sigmoid sinus to the posterior inferior portion of the middle ear, as described in the operation, is found entirely healed. Slitting the sigmoid sinus along its course from the torcular herophili to the jugular bulb, I found it absolutely normal. The anastomosis is seen between the colored silk threads for demonstration purposes, namely, the centripetal end of the hypoglossal has a blue thread, the centrifugal end of the hypoglossal has red thread and the facial stump entering practically at right angles has a yellow thread tied about it.

Dissection of the stylo-mastoid region shows the facial nerve with yellow thread tied through it, demonstrating how very difficult it was to reach during the operation, a fact that caused me to suggest this instrument that can bluntly reach up to the stylo-mastoid foramen.

Of course, a histologic examination of the anastomosed nerves, as well as a more minute examination of the temporal bone, will follow and be of interest. This was impossible to do in order to demonstrate the gross specimen first.

Case II.—Mrs. M. This case has already been reported in the *Laryngoscope* in extenso, on account of the interesting pathologic condition of the temporal bone, so I will just briefly recapitulate in order to make clear the indication for the anastomosis.

She had an endothelioma intravascularis of the middle ear, which destroyed the continuity of structure of the facial nerve, with complete facial paralysis as a consequence. The radical removal of this tumor was not followed by any improvement of this paralysis, but there has been no recurrence of the growth for three years, an interesting fact since these growths are supposed to be sarcomatous.

The patient hesitated for over a year and a half before submitting to the nerve anastomosis. During this time this patient suffered from a general malnutrition and marked anemia which resisted all treatment. In 1907 she decided to have the operation performed, and a test of reaction of degeneration to the facial muscles was plus and a very weak response to the galvanic current, which showed that the muscles were very weak, and if union of the nerves did take place recovery would likely be very slow.

In 1907 the facial hypoglossal operation was performed, Dr. Frank Allport and Dr. Otto Stein, Chicago, assisting. Great difficulty was encountered in finding the facial nerve inasmuch as there had been many scars in the region of the mastoid region, especially low down anteriorly from a very long after-treatment of her first operation. When the facial was finally joined to the hypoglossal, end to side, there was considerable traction in the latter nerve. Again, imbedding this union into the posterior belly of the digastric and closing up the wound. A small granuloma was curetted from the external canal of the left ear, and microscopically proved not to be endotheliomatous.

Patient made an uneventful recovery from the operation, with primary union of the skin incision. This patient made the same remarks, being a more intelligent patient than patient No. 1. She stated that her left cheek felt more natural and stronger. This also disappeared in the course of a week or two. After the patient came out of the anesthetic, I found the anterior and lateral portions of the left side of her tongue paralyzed, none of which was evidenced in Case 1. During the first six months following the operation, during which time the patient received daily electric massage and tonic treatments, there was very little improvement seen; in fact, so far as appearances go, only a beginning crease, such as one sees as a natural contracture following the paralyzed muscles is observed, and only to be differentiated when the patient swallowed. From this time on the improvement was more in the upper branches; she could close her eyes better, and on the application of the faradic current, small contractions followed. However, I want to put this case on record up to the present day, not as a cure, but to prove the point that when one has a positive test of reaction to degeneration, and clinically has strong evidences that the nerve is very likely severed for a greater distance and scars, tumors, sequestra, or other foreign bodies, interposed between its ends, one should do the anastomosis as quickly as possible and not wait for nature to bridge it over. Another point is the general condition of this patient, namely, the anemia.

Case III.—Mr. M. This case has already been reported in connection with the report on Bezold's mastoiditis in *Journal of Ophthalmology and Oto-Laryngology,* 1907, and I will therefore omit the detailed history. Suffice it to say at this time, that the man had a permanent facial paralysis associated with a marked necrosis of the temporal bone along the greater course of the facial nerve, including the nerve. On August 4, 1907, I made a facial-hypoglossal anastomosis, only that the technic differed in making it end to end. Dr. Ray, of Louisville, and Weaver, of Sherman, Texas, assisted. There was again a great deal of difficulty in dissecting the facial, owing to many old scars on the neck from the Bezold mastoiditis many years before. We found quite a number of enlarged lymph glands, submaxillary, over the region of the hypoglossal that required dissection and took considerable extra time.

Observations.—After the patient came out of the anesthetic, he had complete paralysis of the left side of his tongue, and there was considerable difficulty in speaking and swallowing. This man had no such feeling of the paralyzed side of his face as in Cases I and II, after operation—that is, primary tonicity. Ten days after the operation, when the stitches were removed, there was found a small fistula in the region of the parotid gland, discharging saliva, and when the patient would suck a lemon, this flow would be markedly increased. This fistula was very likely produced during the manipulations to dissect the facial nerve by a sharp retractor. This precaution against such an accident is brought out by Murphy.

As to the course of this case, I am not able to report any further than that he left Chicago about one month after the operation, having had no after-treatment. Being a laborer and in very bad circumstances, he went to Cleveland to seek employment. About four months after the operation I received a letter from Dr. Large, who chanced to see him in his clinic, and he states that the face appears completely paralyzed, and the tongue paralysis is giving the patient considerable trouble. No conclusion can be drawn from this report, however, as a test for reaction of degeneration, and the observation of one who has seen the case before operation are necessary.*

Case IV.—Mrs. S., 32 years old. Suffered for a long time with tic doloroux and facial paralysis on the left side. When I first saw her she had received every possible treatment for the cure of her tic, including the injection of osmic acid (Murphy); neurexeresis of the three branches and resection of the Gasserian ganglion (by Carl Beck) without positive relief. Examination of the ear showed no evidences of trouble there, and the test of reaction to degeneration was negative. The eyelids and forehead muscles were not affected. The eye could not rotate outwardly, and there was a paralytic squint. She could not hear in this ear. Therefore, I made a diagnosis of some central pathologic lesion involving the fifth, sixth, seventh and eighth nerves, probably tubercular, as there had been a history of this affection.

On January 7th, 1907, I operated, reopening the original bone flap that had been made by Dr. Carl Beck in operating for the Gasserian ganglion, and found a very markedly thickened dura. (Sectioned later and found just granulations, extradural, but no tuberculosis.) Elevating the brain structure I found considerable matting together of the base of the brain through the dura. I explored the region of the exit of the above nerves from the brain and did not find any gross lesion. Knowing that exploring this region of the brain structure would very likely kill the patient, I stopped the operation. However, before closing up I explored the brain from above in various directions and found nothing in the shape of a tumor. To my astonishment the patient recovered and was free from the neuralgic pains. Enthusiasm on my part and the desire of the patient to have her face straightened, I agreed to do a facial hypoglossal anastomosis. The hypoglossal nerve was of course not involved in the above intracranial trouble. On February 5, 1907, I operated in the usual way, intending to do the end to side procedure. Dr. Vaughan, Chicago, and Dr. Edwards, of Kansas, assisted. When I had the facial dissected and excised from the stylomastoid foramen and attempted to bring it into close proximity to unite with the hypoglossal, I found it impossible. Having just read of Gluck's experiments to use living tissues as conductors for nerve fibers to regenerate, I used a neighboring large branch of the superficial cervical nerves. Excising a long enough piece, I made with it an end to end procedure with the facial, and implanted it into a slit of the hypoglossal as usual. This whole area was covered by a layer of the sterno-cleido-mastoid muscle, that being nearest at hand, to prevent adhesions. Patient recovered from this operation without any untoward symptoms. Shortly after, however, nephritis was diagnosed and a general anasarca followed. The patient did not live more than two months after the performance of this last operation, and consequently I can add nothing further to the subject. There was never any statement made by the patient that she felt any primary tone on the operated side of the face, and no evidences of recovery were demonstrable. The patient had some trouble in swallowing and the tongue lagged considerably on the left side.

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*Just before going to print, patient, Chas. M., presented himself, after an absence of several months (during which time he had been under the care of Dr. Large), showing a complete recovery from his paralysis. Not only has the tone and associated function with swallowing returned, but the patient also has actual voluntary movement of the formerly paralyzed side of the face. This is the first case I have seen to have this phenomenon. I also wish to state that this patient has had no after-treatment, as electricity, etc., and has lived in the poorest hygienic surroundings.

Inasmuch as a post-mortem was not permitted, I cannot say what the conditions of the anastomotic points were, nor can I possibly find out the specific etiologic factor.

Case V.—Mr. W. B. D., Louisville, Ky., aged 53 years. Has always enjoyed good health until he developed an earache in right ear, which was diagnosed as an abscess by Dr. Cheatam, who lanced it and applied leeches, but without relief. Pains did not diminish. He was taken to infirmary after two days' time and drum again incised. Hearing was never much interfered with and there was never much soreness back of ear. Deep-seated pain over right eye. Expectant treatment was instituted, consisting or repeated irrigation of the ear with hot water and lying on hot water bag; this was continued for six weeks, when treatment was changed to flaxseed poultice every 20 minutes for about 72 hours. After elapse of one day Dr. Cheatam performed mastoid operation, following which there was no relief from pain. Six days later second operation was performed by Dr. Cheatam, assisted by Dr. Ray, following which the patient still was not relieved from pain and eye aching. Immediately after recovery from anesthesia facial paralysis was observed.

Findings at time of operation given by Dr. Cheatam and Dr. Ray are as follows:

These deep-seated head and eye pains continued for about three months, gradually abating until they disappeared. The healing of wound back of ear required a considerable length of time, about five months. The discharge from the middle ear ceased a week or ten days after, never recurring. The facial paralysis had never taken on any change in spite of all local and general treatment. Besides difficulty in speech and cosmetic appearance there was a metallic taste in the anterior and lateral half of tongue (right side), which was constant, especially when he cleared his throat. Lacrymation considerable. He did not use right side of face for chewing, as the food lodged in the cheek cavity. Patient thought that salivary secretion and taste might have diminished some. Olfaction did not seem to have been affected. Breathing (nasal) as good as ever. General health has been good except he had some trouble with bladder with bloody urine, which would clear up and return. There was some difficulty in urination, sometimes burning. Examination of urine was made by Dr. Wendell, Dr. Wathen, Jr., Dr. H. Koehler, and kidneys found normal, but there had been no general examination made since operation. Appetite good, patient sleeps well, bowels regular. Habits are, practically abstainer.

Examination.—Characteristic facial paralysis. There was primary contracture at lower lip extending to the margin of lower jaw. Lower lid drooped so as to evert lacrymal duct down and out, showing accumulation of tears constantly in lower sac. The alae of nose, right side drooping, narrowing nostril. Upper lip hung over margin of upper teeth, and lower lip, caused by upper lip, held closer to lip margin. At back of the ear was found evidence of an incision leading from a line drawn across the upper margin of auricle downwards to about an eighth of an inch from the tip of lobule, in middle of which there was a depression in the mastoid process of the size of a lima bean. In the upper margin of incision there was a keloid scar firmly adherent to bone.

Patient was observed last October under superficial examination, but a positive examination made as to a reaction degeneration, which herewith is repeated:

Test of Reaction of Degeneration.—No response to the faradic current. When galvanic current is applied patient feels contractions at make and break, but not observed by myself.

Nasal Examination.—Practically negative with a few irregularities.

Examination of Tongue.—Perfectly straight.

Examination of Palate.—Shows absolute movement normal, the muscle of uvula reacting promptly.

Examination of Right Ear.—Stenotic canal (externally). The tympanic membrane seems to be entirely regenerated. No evidence of disease in remaining portion of canal.

Examination of Hearing.—Hears highest tones. Hears low tuning fork. Weber laterates to right. Rinne negative. Shorter on right side. Rinne positive on left side.

General Examination.—Revealed no pathologic condition as to his kidneys, gastro-intestinal tract and heart and lungs. February 12, 1908, facial-hypoglossal anastomosis was performed, Dr. Ewing M. Day, Pittsburgh, Dr. J. Pratt, Shelbyville, Ky., Dr. L. J. Hughes, of Chicago, assisting. I found the facial nerve unusually easy in this case, for in the former cases this procedure lasted very long. On account of the long continued drainage after the mastoid operation, I expected much trouble from adhesions. We made the end to side implanation and buried the nerve in the posterior belly of digastric. Time of operation 2 hours and 48 minutes.

Observations.—With the exception of a marked nausea, which he said he had also with his other operations, the patient recovered very well and continued to remain normal for five days, when his temperature rose to 103.4°. Examination of the wound showed a marked swelling in the region of the parotid and along the lower jaw. Releasing some of the upper sutures a flow of saliva of established (salivary fistula), and on the following morning he had a temperature of 99°. Every day following until this day, February 21, 1908, his temperature fluctuated between 99° and 102°, with some degree of uncomfortable feeling. Examination of the pharynx shows a small degree of edema of the uvula. There can be no further statements made at this early time as to results.

CONCLUSIONS.

The fact that one case out of a dozen of facial-hypoglossal anastomosis will recover is sufficient encouragement and indication to justify its performance.

In order not to bring this procedure into disfavor, one should select his cases and this selection should be based upon a thorough examination, especially as to the reaction of degeneration. That is to say, the muscles must be present, no matter how flaccid, and the galvanic electric current will bring that out, as stated above.

A thorough knowledge of the anatomy and technic is absolutely necessary and I know of nothing better to recommend than practice suturing of nerves on dogs and perform the operation on the cadaver two or three times before doing the operation.

One should not be satisfied by simply doing the operation, but great care should be exercised in the long, continuous after-treatment of electricity, massage, etc.

Since the reading of this paper, the author has operated on the sixth case which has the following history:

Mr. B., 18 years of age, referred to me by Dr. Casey Wood, when 1½ years old had a marked mastoid disease with secondary abscesses in the neck, anterior and posterior, as well as anterior to the auricle in the parotid regions of the left side. Dr. Barkan, of San Francisco, operated at the time, and it required a great length of time before boy recovered. His face, previous to the mastoid operation, was straight. Following the procedure he had complete facial paralysis.

Examination.—Characteristic picture of the condition with all the usual symptoms. A number of large scars are found on his face, neck and back of the ear. In this locality is quite a large fistula, communicating with the auditory canal, but it is entirely dry. Test of reaction to degeneration shows it is negative, that is, there is no response to any kind of current, and consequently a case with very poor prospects of recovery. In fact, from the length of time that the paralysis has existed one would not expect any result and is a contraindication to the operation.

I wish, however, to state, that as this is the son of well-to-do parents, who took advantage of everything known to them to aid the boy in recovering function in the paralyzed side of his face, the patient has, all these years had stimulating treatments to the nerves and muscles of the face. Consequently there was some chance that remnants of muscles might be present.

Dr. Casey Wood and the father decided, nevertheless, to have me try to do an anastomosis and take a chance on the result.

Operation.—Through one of the existing neck scars I soon reached the region of the facial nerve. To my great disappointment I found the complete absence of any such structure, and, if it was present at all, it was not recognizable as a facial nerve, nothing resembling a nerve structure in the whole course. I dissected towards the face and here also found no trace of any of the branches. Consequently I closed up the wound and stated to the parents the absence of the facial nerve. The young man made an uneventful recovery from the operation.

Two weeks later I performed a plastic on his face in the following manner: Through a scar situated over the zygomatic region which was first resected, a long straight needle armed with strong silk was passed well under the skin towards the labial fold, there made a loop, and needle again made to come through the original entrance opening. Drawing the thread taut, somewhat puckering the skin so as to make an artificial labial fold, the threads are tied and the wound closed by primary intention. Patient recovered with a very good cosmetic result, as the accompanying pictures show.

Case IV.—Mr. M. has reported since the reading of the paper and sent a photograph. It shows distinctly a formation of labial fold and he reports associated movements of his face with the act of swallowing. The tone of the paralyzed side has much improved.

Case V.—Mr. D. Doctor Moren, of Louisville, reports that the muscles of his paralyzed side of his face are responding much better and patient declares an improvement in the tonicity of his face.

Case II.—Mrs. M. has an improvement in her facial expression but her general condition of anemia is constantly increasing and the chances of recovery are not very good.

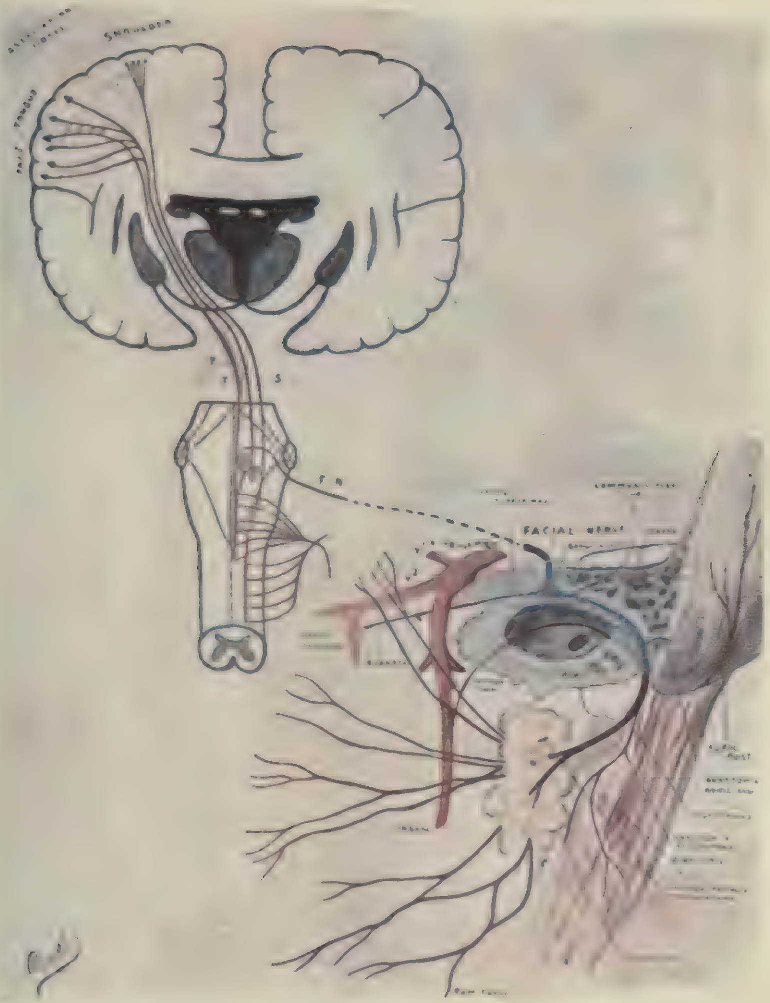

FIG. 1

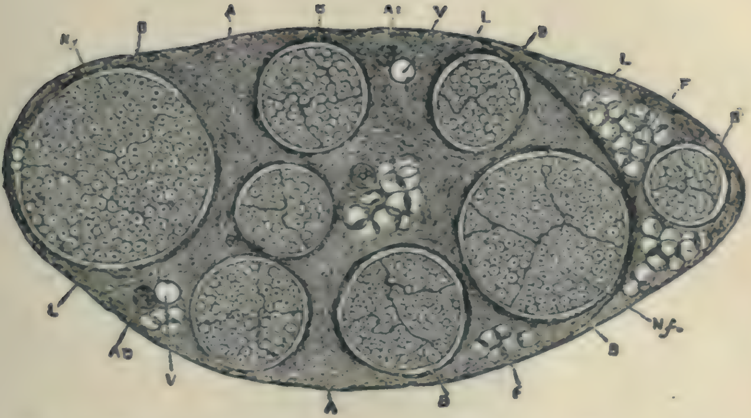

Fig. 2.—Transverse section of the sciatic nerve of a cat about x 100.—It consists of bundles (Funiculi) of nerve-fibres ensheathed in a fibrous supporting capsule, epineurium, A; each bundle has a special sheath (not sufficiently marked out from the epineurium in the figure) or perineurium B; the nerve-fibres Nf are separated from one another by endoneurium; L, lymph spaces; Ar, artery; V, vein; F, fat. Somewhat diagrammatic. (V. D. Harris.)

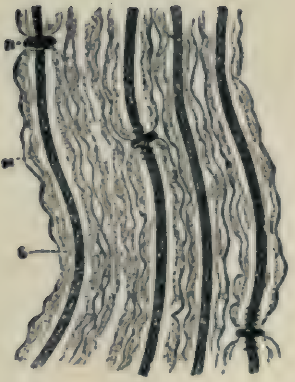

Fig. 3.—Several fibres of a bundle of medullated nerve-fibres acted upon by silver nitrate to show peculiar behavior of nodes of Ranvier, N, toward this reagent. The silver has penetrated at the nodes, and has stained the axis-cylinder, M, for a short distance. S, the white substance. (Klein and Noble Smith.)

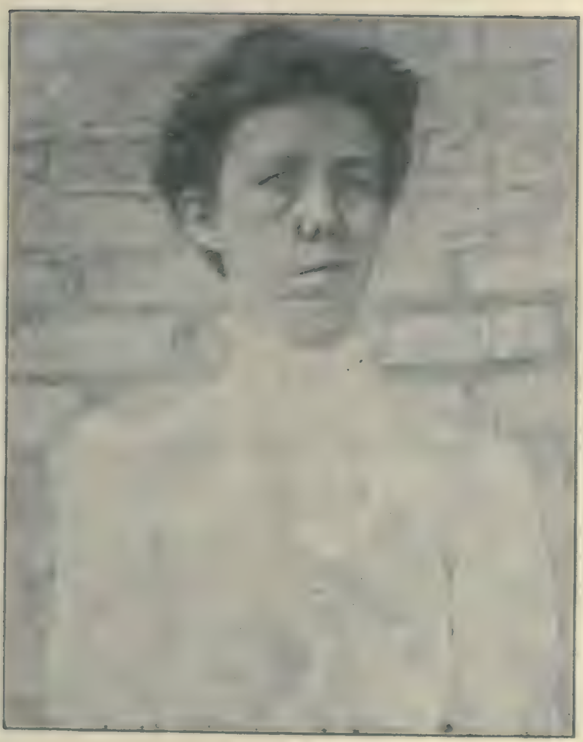

Fig 4.

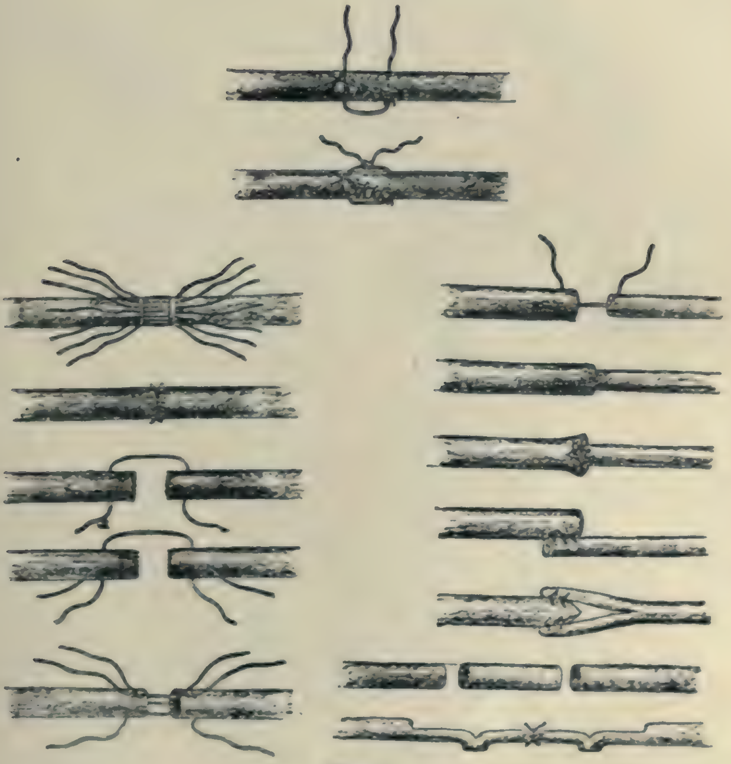

Fig. 5.

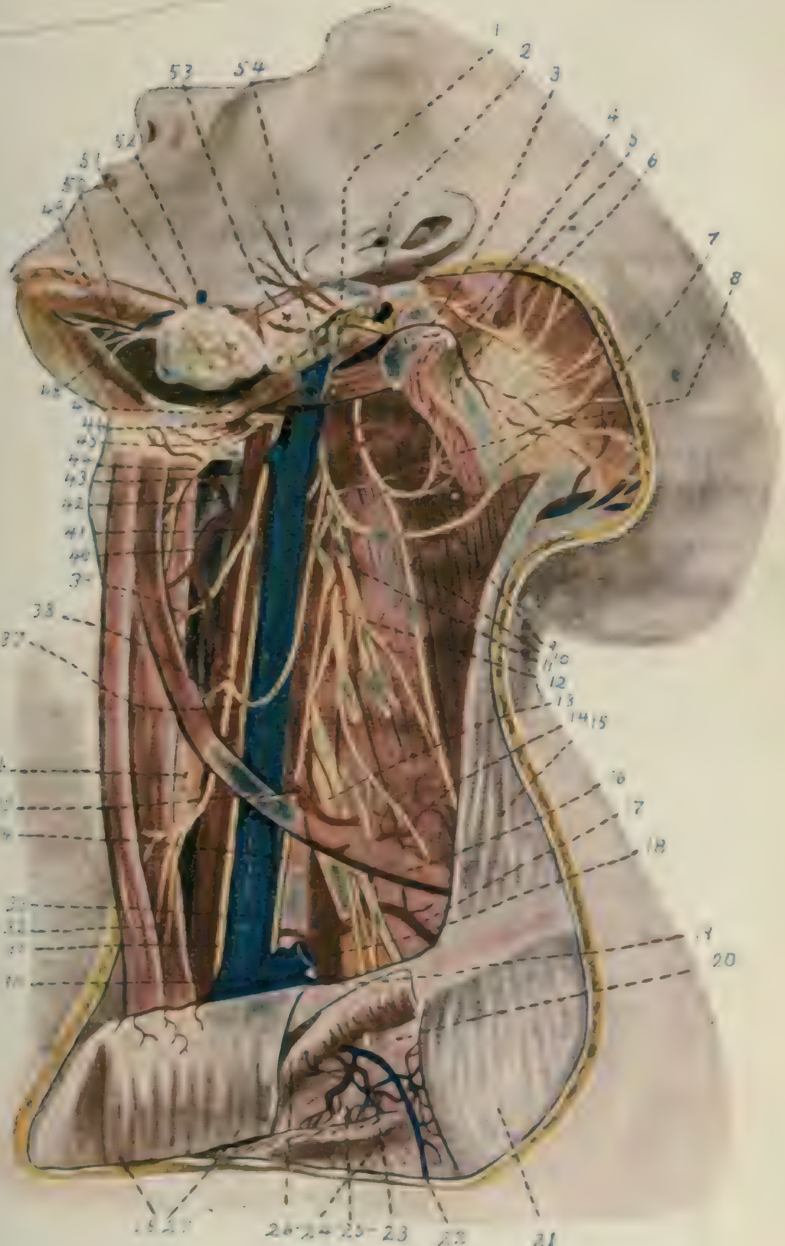

FIG. 6.
See next page.

Fig. 6.

Fig. 6—Represents the surgical anatomy of the field of operation of the facial nerve with the other two nerves (spinal accessory and hypoglansal).

1. Sternocleidomastoid artery.
2. Facial nerve.
3. Occipital branch of posterior auricular artery.
4. Posterior auricular branch of facial nerve.
5. Mastoid tip.
6. Sterno mastoid, muscle cut and retracted.
7. Lesser occipital nerve.
8. ii Cervical nerve (ventral Br).
9 Accessory nerve.
10. Accessory nerve.
11. iii Cervical nerve (ventral br).
12. iv Cervical nerve (ventral br).
13. Brachial plexus.
14. Ascending branch of transverse cervical.
15. Trapezius.
16. Omohyoidens.
17. Descending branch of transverse cervical artery.
18. Subclavian artery.
19. Subclavian vein.
20. Pectoralis minor.
21. Deltoidens.
22. Cephlic vein.
23. Thoracs acromial vessels.
24. Clavicular portion of pectoralis major.
25. Ant. Thoracic nerve.
26. Clavacular port. of pectoralis major.
27. Sternocleidomastoidens.
28. Sternocleidomastoidens.
29. Sternocleidomastoidens.
30. Innominate vein.
31. Vagus nerve.
32. Com. cartoid nerve.
33. Inf. bulb of int. jugular vein.
34. Phrenic nerve.
35. Super. cervical artery.
36. Thyroid glands.
37. Ansa Hypoglossi.
38. Descending br. of hypoglossi.
39. Ascending br. of cervical artery.
40. Superior thyroid artery.
41 Descending br. of hypoglossal.
42. Superior laryngeal artery.
43. Ext. caroted artery.
44. Thyreohyoid br. of hypogl.
45. Lingual artery.
46. Hypoglossal nerve.
47. Stylohyoidens.
48. Digastricus.
49. Submental artery.
50. Mylohyoid nerve.
51. Submaxillary gland.
52. Ant. facial vein.
53. Paroted gland.
54. Posterior facial vein.

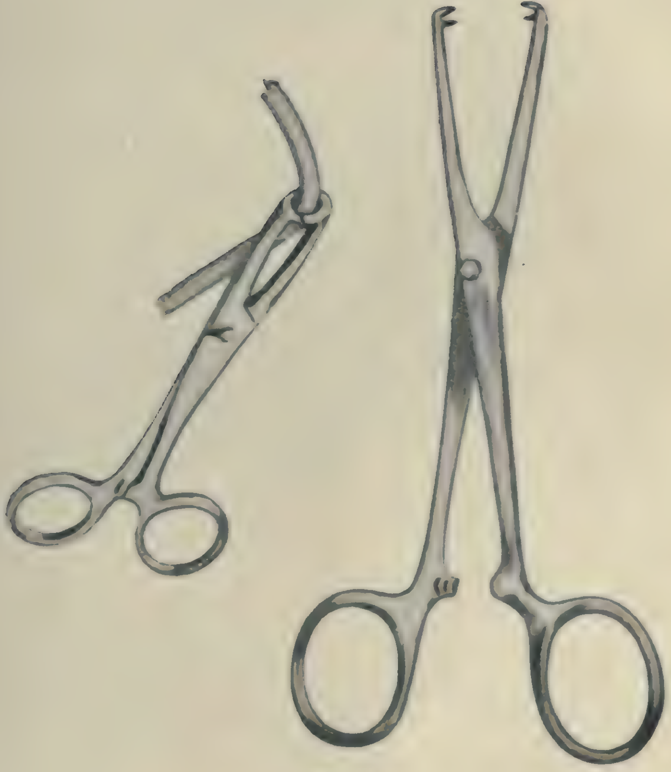

Fig. 7.

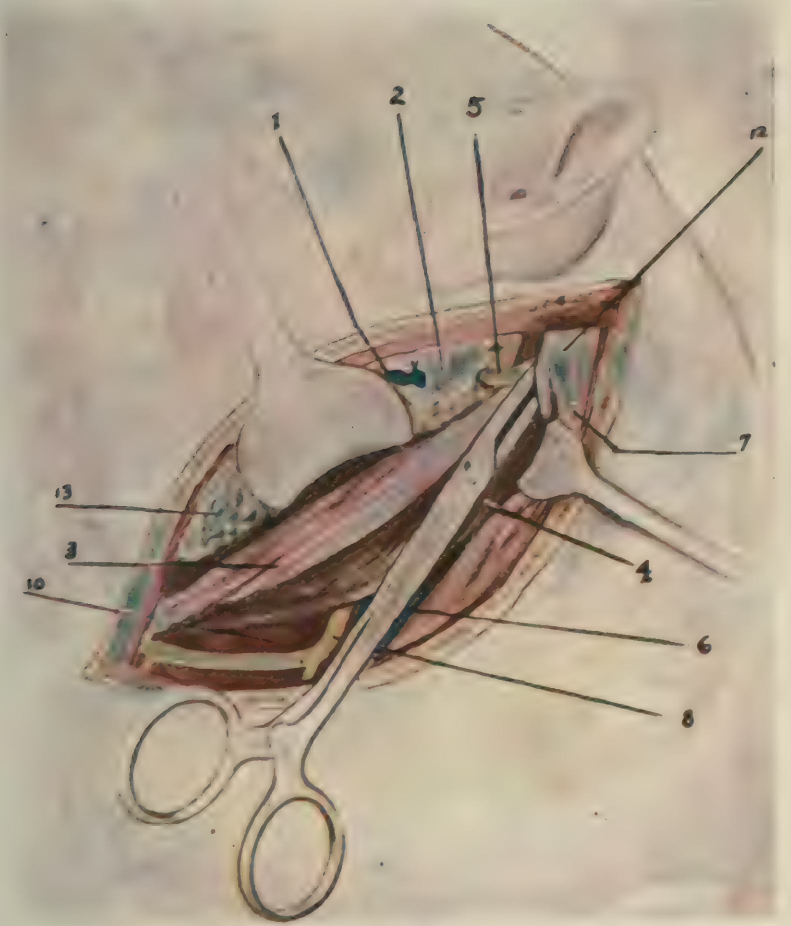

Fig. 8.

Illustrating the instruments in place for dissecting the facial nerve into the depths of the stylomastoid foramen.

1.	Ant. facisl vein.	6.	Int. jugular vein.
2.	Parotid gland.	7.	Sternocleidomastoidens.
3.	Stylohyoidens.	8.	Hypoglossal nerve.
4.	Post. belly of digastricus.	10.	Common caroted artery.
5.	Facial nerve cut.	12-	Mastoid tip.
		13.	Submaxillary gland.

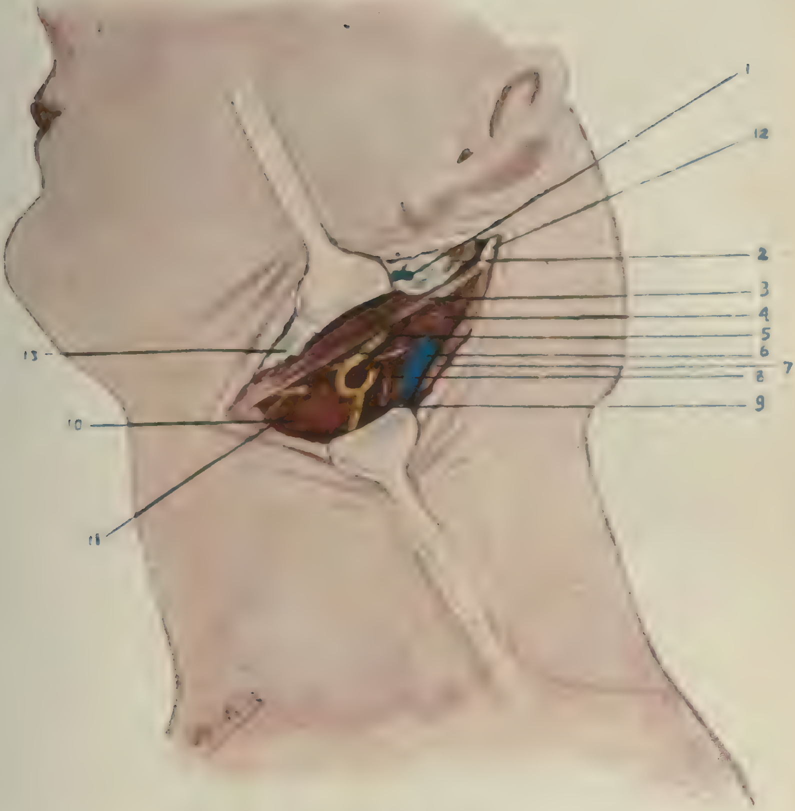

Fig. 9.

Fig. 9. Representing the facial hypoglossal anastomosis by the end to end method.

1. Anter. facial vein.

12. Mastoid tip.

2. Parotid gland.

3. Stylohyoideus muscle.

4. Post. belly of digastric M.

5. Facial nerve (cut off at stylomastoid foramen and turned down under stylo-hyoideus, implanted into No. 11).

6. Internal jugular vein.

7. Sternocleidomastoideus muscle.

8. Hypoglossal nerve.

9. Hypoglossal nerve, its descending branch.

11. Hypoglossal nerve (with the facial stump implanted).

10. Common carotid artery and its subdivisions.

13. Submaxillary gland.

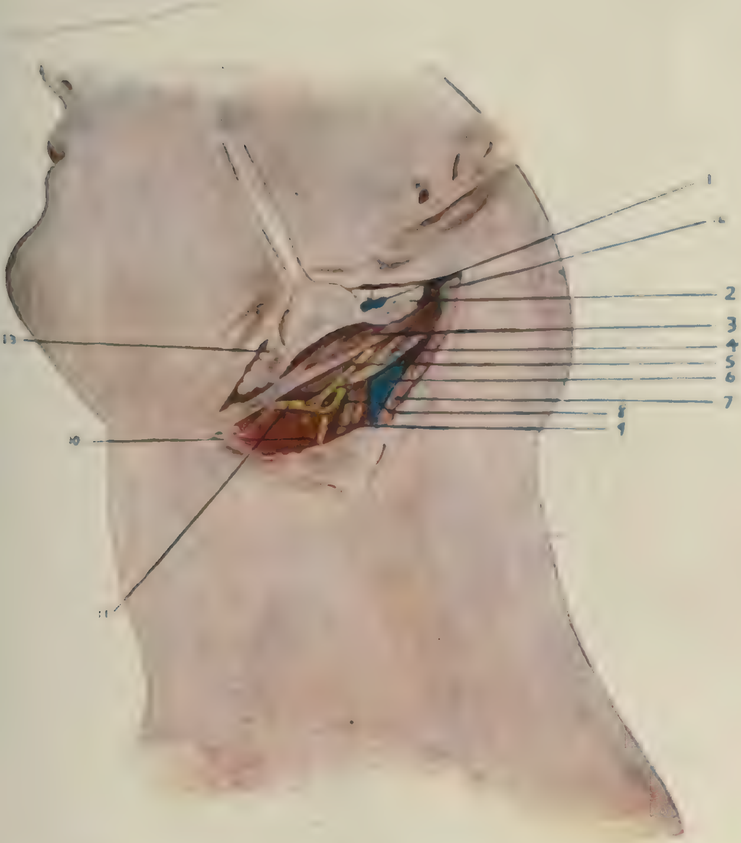

FIG. 10.

Representing the facial hypoglossal anastomosis by the end to side method.

1. Ant. facial vein.
2. Parotid gland.
3. Stylohyoidens.
4. Post. belly of digastricus.
5. Facial nerve cut and pulled under the stylohyoidens.
6. Internal jugular vein.
7. Sternocleidomastoidens.
8. Hypoglossal nerve.
9. Hypoglossal nerve, descending branch.
10. Common carot id artery.
11. Hypoglassal nerve.
12. Mastoid tip.
13. Submaxillary gland.

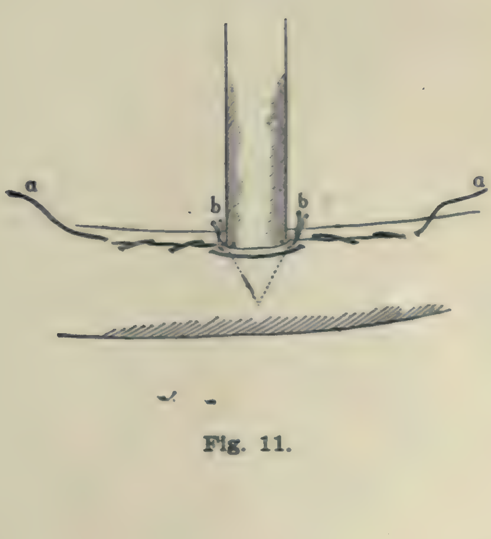

Fig. 11.

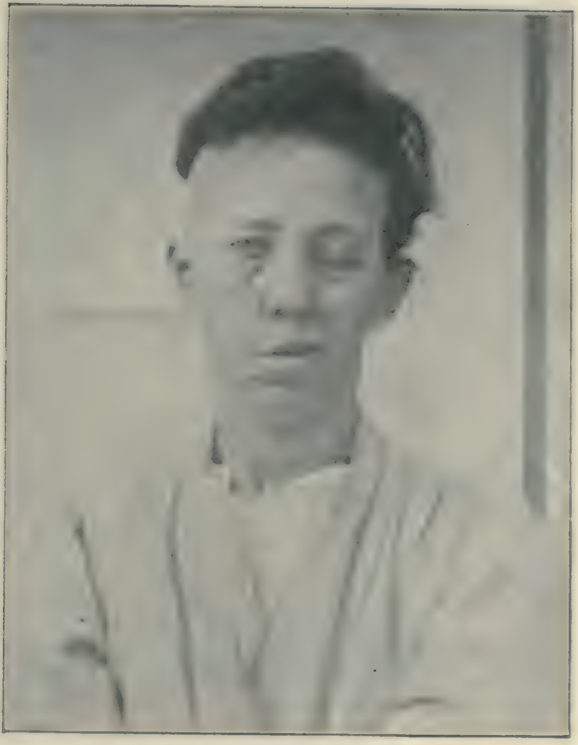

Fig. 12.

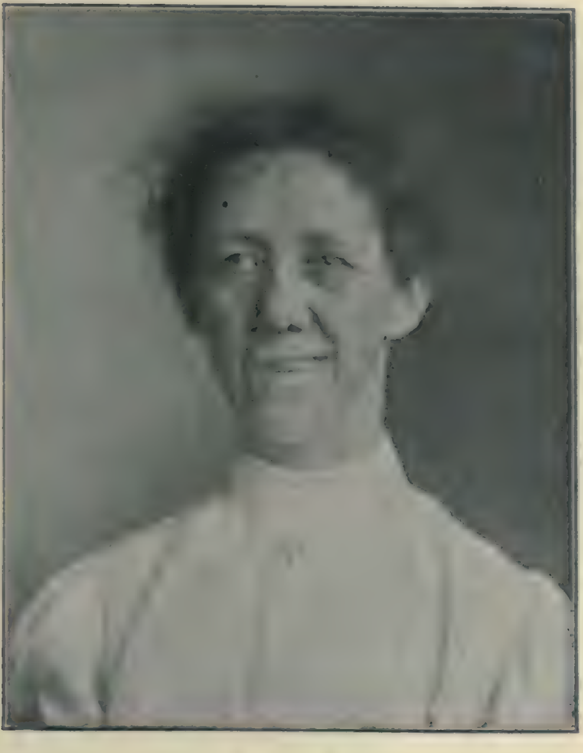

Fig. 13.

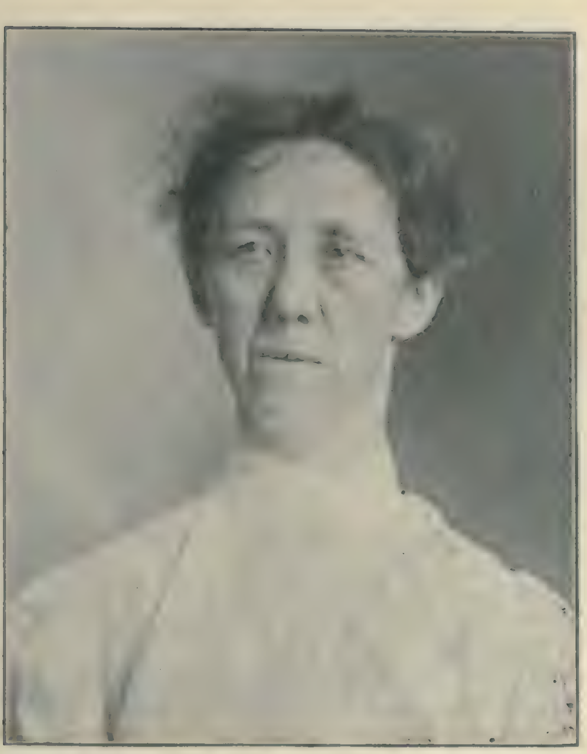

Fig. 14.

X.

THE OPERATION FOR THROMBOSIS OF THE SIGMOID SINUS AND INTERNAL JUGULAR VEIN, OF OTITIC ORIGIN.*

By Frank Allport, M. D.,

Chicago.

One of the most important sequelae of middle ear infections is phlebitis or thrombosis of some of the venous drains of the head, and particularly when such pathologic conditions occur, as they usually do, in the sigmoid bend of the lateral sinus. Disease of this nature is found not only in the sigmoid sinus, but also in the lateral sinus proper, the superior and inferior petrosal sinuses, the cavernous sinus, the jugular bulb, the internal jugular vein and its connections, etc. The sigmoid sinus is, however, its favorite location on account of its vascular connections with the tympanic cavity and adjoining cells and its close contiguity to these frequently infected structures. As the jugular bulb is located directly beneath the tympanic cavity, an unusually high dome to the bulb, or necrosis of the tympanic floor, or congenital dehiscences in the floor, will assist in the conveyance of infections. This will occur through the vessels and lymphatics connecting the tympanic cavity with the bulb, and may produce a primary phlebitis or thrombosis of the bulb, which has not proceeded through the usual channels of the antrum, mastoid cells and lateral sinus. These conditions are usually found in children, develop rapidly, and generally demand immediate operation with excision of the internal jugular vein. I do not believe that actual intravascular phlebitis and thrombosis of the sinus and vein occur as frequently as might be supposed from a perusal of much of the available literature of the day, because a fairly large experience in mastoid work for many years has not disclosed this to be the case. Nevertheless, the disease occurs with sufficient frequency to demand the most careful study in order that prompt and skillful action may be taken whenever its existence is actually disclosed.

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*Read before the Middle Section of the American Laryngological, Rhinological and Otological Society, Indianapolis, February 22, 1908. From the Journal of the A. M. A.

RISK IN SINUS OPERATIONS.

The surgery of otitic phlebitis and thrombosis has made great progress of late years and many lives have been saved thereby that must otherwise have been inevitably lost. I fear, however, that over-enthusiasm along these lines may have led some thoroughly competent and trustworthy observers to overestimate the frequency and importance of the subject and to underestimate the dangers of operative interference. The opening of the sigmoid sinus, for instance (first seriously proposed by Zaufal in 1880), with its possibilities for infection, especially when done through an already infected mastoid cavity (which is, of course, usually the case), is an operative procedure of importance and danger. Yet some surgeons practically advocate its performance for diagnostic purposes where any reasonable suspicion points to an intrasinus involvement. In my opinion the sinus should usually be opened in the face of mastoid disease, either acute or chronic, accompanied by characteristic phlebitic symptoms, such as chills, sweats and wide excursions of temperature and pulse. It should also be done in the presence of a virulent infection, especially of the streptococcus type, a significant differential blood test, etc. Nevertheless, I have seen a number of such cases cured by the removal of necrosed bone, granulations[1] and pus from the sinus neighborhood. I should, therefore, be inclined to wait a few hours after the removal of such débris, before opening the sinus itself, unless the patient's condition and symptoms are reasonably decisive and urgent in character, and unless an inspection of the exposed sinus renders the presence of a thrombus exceedingly probable.

It must have been the experience of most surgeons that phlebitic symptoms frequently and permanently may be relieved by a removal of neighboring necrosis, granulations, pus, etc., which perpetually infect and reinfect the sinus walls. This fact is especially emphasized by Bergman in his writings and can certainly be substantiated by all operators of experience. In this observation lies an earnest warning against too energetic operative procedures, without quite positive indications. It is sometimes the case that septic symptoms following a mastoid operation are due to pneumonia, typhoid, malaria and other coincident conditions which should be duly considered and properly weighed before proceeding to a sinus operation. It should not be forgotten that, while lives may occasionally be sacrificed by proper conservatism, it is probable that more lives would be lost by injudicious and hasty surgery.

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[1] The existence of granulations on the soft sinus wall is regarded by many as beneficial, in that it is nature's obstruction to the invasion of microorganisms, and many surgeons carefully refrain from disturbing them during the course of an operation.

The provisional determination to open the sigmoid sinus is usually formed before the mastoid has been operated on, or after an operation has been performed and several days have perhaps elapsed, during which time the condition of the patient has not materially improved. This resolution is, of course, liable to be changed by existing intramastoid conditions, after the mastoid cortex has been removed, at the time of the primary operation. In all cases where sinus symptoms are clearly present, the bony covering should be freely and carefully removed and the soft sinus exposed for inspection, whether the osseous partition is necrosed or not. Should the inspection reveal an apparently healthy sinus, operative procedures may be suspended (unless the patient's condition is too grave), as sharp sinus symptoms are sometimes produced by a beginning absorption or phlebitis. This may originate in an infected mastoid cavity, relief from which is not infrequently noticed after a thorough exposure and cleaning of this space.

After this has been accomplished and the patient carefully watched for some hours or perhaps one, two or three days, and the sinus symptoms remain the same or grow worse, the sinus should be unhesitatingly opened, even if its local appearances are apparently healthy. Much the same procedures should obtain in case necrosis, granulations and pus are found in the sinus region, for such pathologic products continually bathing the soft tissue tend to become absorbed and to produce the characteristic danger signals. Such debris may be carefully removed and the sinus amply inspected. Should it be found of healthy appearance, one may stay operative procedures a little, unless the symptoms are quite urgent. Many patients have recovered and will recover under such conservative management, and this fact points a significant lesson.

THE APPEARANCE OF THE UNCOVERED SINUS.

Of course, the entire subject of opening the sinus hinges on the probable existence or non-existence of a thrombus, and this cannot always be easily determined. A discussion of this topic is too prolonged for the present article, and is apart from the specific subject under consideration.[2] As the physical appearance of the uncovered sinus, however, must be observed while operating on a patient suffering with sinus symptoms, this phase of the subject may with propriety be here considered, and it may be said that even a physical examination of the sinus does not always clear up the pathology. The sinus is usually bluish-white in color and easily compressible. Nevertheless, the neighboring necrotic bone, etc., may stain and darken the outer coats of the vessel, without the existence of any intramural clot, or a perfectly normal color may characterize a sinus, in whose interior a thrombus may be found.

A sinus may be thoroughly compressible and yet contain a parietal clot, where the obstruction is not yet complete, but a solid, resisting sinus is, of course, a reasonably positive sign of an obstructive clot. The emission of pus from the mastoid foramen is a significant symptom, as it may then be assumed that extradural pus is present in the cerebellar fossa, around the sigmoid groove. I am opposed to the insertion of an aspirating needle into the sinus in order to ascertain the fluidity of its contents. If nothing is withdrawn it is safe to assume the presence of a thrombus, but blood may escape in case of a partially organized clot, and if the vessel is healthy the operator may easily regret having punctured and probably infected a normal sinus; if the sinus is to be opened at all, it is much better to make a complete incision rather than a puncture. The stroking and compression test of Whiting cannot be commended, for, while it may facilitate the diagnosis, it can easily displace infected thrombotic particles and set them into circulation with disastrous results.

THE PROBLEM OF DIAGNOSIS.

It will thus be seen that making a diagnosis in cases of otitic sinus thrombosis is beset with some difficulties and perplexities. Yet a correct conclusion can usually be reached, especially when a consideration of the general subjective and objective symptoms has been aided by an operative exposure of the sinus itself through the mastoid cavity. Fortunately this (mastoid operation) is a procedure which should be performed primarily and, therefore, nothing is lost and everything gained when a mastoid operation is advised in a suspected otitic sinus thrombosis, for the cells, etc., may be thoroughly opened and cleaned and the sinus inspected and perhaps opened during one operative procedure.

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[2] A tolerably complete analysis of the symptomatology of sinus thrombosis, etc., can be found in a communication by the writer in the Chicago Medical Recorder, January, 1906, and in other books and periodicals.

Should an operation, or at least an inspection of the sigmoid sinus be decided on, in a case of otitic origin, it is only necessary to consider the operative steps in connection with a thorough exposure of the mastoid cavity, for this is at the same time the easiest as well as the best avenue of approach. The mastoid operation should first be completed in all its details, for, in case troublesome hemorrhage occurs, it might be extremely difficult to continue further operative procedures on the bone. In addition, the work on the sinus should be prosecuted under the most aseptic conditions possible, and this can be best accomplished after the mastoid interior has been thoroughly cleaned. As has been previously said, the bony covering of the sinus may be in a necrotic, softened and fistulous condition, surrounded by granulations and bathed with pus.

THE REMOVAL OF GRANULATIONS.

The removal of such pathologic products can be accomplished with comparative ease by curette, chisel, bur or rongeur, extreme care being exercised in all such manipulations to avoid injuring the soft dural covering of the vessel. Some authors advocate the non-removal of granulations, believing them to be Nature's obstruction to the invasion of the sinus interior by microorganisms. However this may be in theory, I believe that such tissue is best thoroughly removed, not only to place the parts in as clean a condition as possible, but also to facilitate a careful examination of the sinus, which cannot be satisfactorily accomplished through a granular mass. In most instances I accomplish this work by means of a strong, narrow rongeur, one blade of which is inserted inside and the other outside of the bony sulcus, being careful to avoid pressure on the underlying vessel and only removing small fragments of bone at a time. Objections have been made that the slight pressure on the sinus may induce a fracture of its possibly thrombotic contents, which will be swept into the circulation and induce migratory infection. This objection seems like a refinement of technic and a magnifying of possibilities, for, while such an accident might possibly occur, it is altogether unlikely. It certainly would seem to be a better instrument than the chisel, with its possibilities of bone fracturing and splintering, with perhaps a sinus puncture and an embarrassing hemorrhage.

All the necrosed bone should be removed, no matter how far it extends, either in the direction of the torcular or the bulb, and it is well to remove even some healthy bone in order to insure complete elimination of all necrosis (Fig. 1). The sinus should then be minutely inspected, and if its opening is determined on the operation should be continued after the parts have been thoroughly cleaned and irrigated with antiseptic solution. If it is deemed best to await developments the wound should be cleaned, irrigated and dressed as after other mastoid operations.

In case a primary opening of the sinus is determined on either at the original mastoid operation or at a later date, it should be remembered that an ample exposure is absolutely essential to correct surgical manipulations. The sinus must be exposed, therefore, for at least a short distance above its knee and as far downward in the direction of the jugular bulb as possible. This free exposure may be accomplished by a removal of the necrosed bone alone, where such necrosis is extensive in character, or it may be necessary to remove considerable healthy bone in order to render the sinus easily accessible.

LOCATION OF THE SINUS.

Unless the situation of the sinus is indicated by a fistula, its position is uncertain. In children it is located more superficially and further forward than in adults, and even in the latter its location is by no means fixed. It is usually, however, to be found at a point about one-half an inch behind the posterior bony meatal wall, just back of the antrum, and a thorough mastoid operation will usually disclose its convexed outlines. The sinus should first be exposed by chisel, bur or curette, after which the bone is best removed with a strong, delicate and narrow rongeur, as previously described. The bone should be carefully removed in small pieces and the operative field examined after each bite of the forceps, care being taken not to wound the sinus, either by perforating its covering by a sharp fragment of bone or by rupturing an already softened dura by carelessly pulling away adherent osseous chips. The latter accident may be averted by, from time to time, passing a small spatula between the soft sinus and its bony covering and gently separating the two.

After the sinus has been fully uncovered all debris, etc., should be thoroughly removed and the operative field abundantly flushed with antiseptic. The hands and instruments of the operator should be recleaned and every effort made to prevent sinus infection. In opening the sinus there is danger of a fatal aspiration of air occurring, to prevent which the patient's head should be dropped low and the operative area flooded with sterile water. The sinus should now be opened by a free incision along its length, being careful not to pass through both walls of the sinus into the cranial cavity, an accident which is by no means impossible. If there is a flow of blood, two tight rolls of gauze or cotton held in fixation forceps (which should be previously prepared) should be placed one above and the other below the incision, remembering that brain substance and not bone lies beneath the sinus, and that the amount of pressure should, therefore, be limited (Fig. 2). Such pads may be held by the operator, or by his assistant, or both operator and assistant may each hold one; they should not be applied, however, until after the sinus has been opened and an opportunity has been given for a burst of hemorrhage to occur and possible thrombotic particles to become expelled. These pads may be lifted alternately and accurate information obtained as to whether the hemorrhage occurs from above or below.

If free hemorrhage occurs from both directions it is proper to assume the absence of a clot, or at least that any existing loose clots have been expelled by the force of the escaping blood. If the hemorrhage is slight or absent from either direction, a clot in that direction is to be assumed. If the vessel is assumed to be healthy, some iodoform gauze, rolled into a cylinder long enough to cover completely the sinus incision, should be firmly placed over the vessel and supplemented by layer after layer of gauze carefully held in place. The other portions of the mastoid cavity should then be packed with gauze and the entire mass of packing held in position by a firmly applied bandage which may usually remain in position for three, four or five days, when it should be carefully soaked off (to prevent hemorrhage) and more packing and bandage applied.

The danger of hemorrhage is usually past by the time the second or third dressing is done. In case there is hemorrhage from below and not from above, the sinus should be further incised in the direction of the torcular, and more of the bony sulcus removed if necessary, in order that adequate room for further operative procedures should be afforded. In considering thrombi, which extend from the sigmoid sinus back toward the torcular, it should not be forgotten that clots extending in that direction are not usually so infective in character as those spreading in the direction of the bulb. The venous current, of course, naturally flows from the head downward, and, while infectious particles may easily pass down with the blood current, they do not so easily pass upward and backward against the current. It should not be forgotten, however, that blood currents in valveless veins seek the direction of least resistance and may easily become reversed on contact with intravenous obstructions. It is, therefore, quite possible that, while a thrombus in the immediate vicinity of the antrum may be highly infectious in character, as it extends backward toward the torcular it may gradually become perfectly innocent, and composed of benign accretions of normal blood clots, that form a natural and healthy obstruction to the passage of infectious microorganisms.

TREATMENT OF THE THROMBUS.

It is, therefore, questionable just how much such a thrombus should be disturbed, and some authors strongly advocate leaving it alone. I believe that if the clot is hard and apparently extensive, it should be gently curetted at its visible portion, in order to remove its infectious layers, and then sprinkled and packed with iodoform powder and gauze. In case, however, the clot is small, or soft and unhealthy in its appearance, it should be completely removed by the curette until free hemorrhage occurs (it being remembered that quite free hemorrhage may occur, even with a considerable parietal thrombus, or from, for instance, the superior petrosal sinus, though the sigmoid sinus is more or less blocked with thrombotic accretions) even if it becomes necessary to remove more and more of the bony sulcus with a corresponding extension of the sinus incision. A small-sized curette should be employed for the removal of thrombi, small enough to allow of intrasinus manipulations, and its spoon may, of course, be carried considerably beyond the limits of the bony opening (Fig. 3).

It should not be forgotten that a sinus infection of several days' duration may soften the sinus walls to such an extent as to render their perforation by a curette exceedingly easy; curetting should, therefore, be carefully performed, lest brain infection be thus produced. Ease of manipulation may be much facilitated in all sinus curettements by not only incising the sinus, but by excising a large piece of its outer dural wall, and diagnosis and surgical precision will be greatly aided by exposing the intrasinus openings of the mastoid emissary vein and the superior petrosal sinus. The former will be found in the posterior wall of the sinus at a point about on a level with the floor of the ordinary radical mastoid operation when finished. The latter enters the sigmoid sinus from the cavernous sinus at a point about on a level with the roof and posterior wall of the ordinary radical mastoid operation, when completed; in other words, at about the junction of the lateral with the sigmoid sinus, and passes into the sigmoid sinus.

In endeavoring to get as close to the jugular bulb as possible the surgeon should not press too far forward, as he is liable to injure the facial nerve, as it passes down through the posterior meatal wall on its way to the stylomastoid foramen. Bleeding may easily be stopped by carefully applied gauze rolls. In case free hemorrhage does not proceed from the bulbar direction of the sinus, a very serious condition confronts the operator, for, the blood current of the inferior petrosal sinus entering the anterior portion of the jugular bulb, on its way from the cavernous sinus, constantly playing on the septic end of the thrombus, naturally carries the infectious microorganisms down into the bulb, internal jugular vein, etc., toward the heart, lungs, liver, etc., thus readily producing sepsis, pyemia, metastatic abscess, etc., which usually result fatally.

It is, therefore, necessary that this end of a thrombus, or a thrombus located in this portion of the sinus, should be as thoroughly removed as possible, in order that the further or general spread of infection may be stopped. This may probably be best accomplished by the curette, which should be gently manipulated until free hemorrhage from below occurs, which may be controlled by the rolls of gauze, bandage, etc., as previously described. It must not be forgotten, however, that it is usually quite impossible to successfully curette the jugular bulb by simply proceeding downward by way of the sinus. The curette may reach practically as far as the bulb and is then stopped by an intervening wall, an abrupt curve, etc., so that if a complete thrombus extends downward as far as the bulb or further, free hemorrhage will not usually be produced by sinus curettement.

Under these circumstances the operation of Grunert, Voss, Iwanoff and others has been proposed for the thorough exposure of the bulb. Owing to the complications and great danger of producing facial paralysis, hazardous hemorrhage, etc., however, it has never become popular and it is generally thought better to expose, ligate and partially remove the internal jugular vein and then clean out the bulb. It must not be forgotten that this does not always prevent the spread of infection by other channels, and that the sudden stoppage of the venous circulation may force infective particles backward into the meningeal and cerebral veins. While the internal jugular vein is, of course, the main avenue through which infection spreads from the sigmoid sinus to the heart and other organs, nevertheless, even with this principal channel ligated, infection may proceed through the posterior condylar vein, the occipital sinus and the mastoid emissary vein, all of which communicate indirectly with the subclavian and innominate veins, irrespective of the internal jugular. It will be remembered that the posterior condylar vein passes through the posterior condylar foramen and connects the lowest level of the sigmoid sinus with the deep veins at the back of the neck.

LIGATION OF THE INTERNAL JUGULAR.

Some surgeons invariably operate on the internal jugular before proceeding to open the sinus; others only ligate when free bleeding cannot be established from the bulbar end of the sinus; others open and curette the sinus and then wait for a continuance or exaggeration of the symptoms, when the jugular operation is performed. Certainly but few, if any, surgeons would sympathize with the views of Horsley, who only ligates after metastases have occurred, for then it would almost surely be too late. Favorable reports of jugular ligation in sinus thrombosis need not necessarily be attributed to the ligation, as, for instance, Körner reports 43 per cent of recoveries without ligation. Still it is probably true that in septic and pyemic cases sinus operations with jugular excision yield better results than when the jugular is not disturbed.

My method of procedure is as follows: The sinus is opened, the head always having been previously lowered, the upper portion is curetted if necessary. If hemorrhage occurs freely from below the operation is finished; if not, the lower end of the sinus is carefully curetted and if free hemorrhage occurs nothing further is done. If free hemorrhage does not occur, pressure pads and a bandage are quickly applied and the jugular is ligated and excised, the bandage then removed, the sinus, bulb, etc., curetted and cleansed and the entire operative field, both in the bone and neck, properly dressed and bandaged. The necessity for the operation is greatly emphasized if the cervical tissues just below the mastoid are swollen and painful, and if any hardness and pain can be detected along the course of the vein in the neck, and if general pyemic symptoms are present.

I am convinced that excision of the jugular is not an operation which should be undertaken hastily, as grave circulatory and other disturbances are liable to ensue, and other methods of relief should first be tried, unless the patient's condition is very urgent. The literature furnishes many instances where sudden optic neuritis, edema of the brain, with hemorrhages and softening, thrombosis of other cerebral sinuses, etc., have followed jugular ligation, and it should not be forgotten that a small and insufficient jugular on the other side, totally unable to assume double duty, may be present and precipitate most unfortunate results. As a deduction from these observations Eagleton urges that in cases of sinus thrombosis, with optic neuritis, it should be concluded that the return venous flow is already seriously obstructed and that great caution should be observed in adding to such conditions by ligation.

EXCISION RATHER THAN LIGATION.

In case it is concluded to operate on the jugular vein it is better to excise most of it rather than merely to apply a ligature, as, if the jugular operation is necessary at all, its septic or pyemic current should be eliminated as much as possible and its poisonous contents removed. Before operating on the jugular the shoulders should be elevated on a sandbag or something of the kind and the neck and head allowed to drop down, the latter turned away from the field of operation in order to place the neck well on a stretch, so that the anatomy of the parts is well defined and the tissues easier to operate on.

If the patient is a male the beard should be shaved and the skin antiseptically cleaned, care being taken not to disturb and break up a possible clot by rough handling. In the case of a male where it is at all suspected that a jugular operation will have to be performed the beard should be shaved before the mastoid operation is commenced. The neck should now be placed on a stretch and the anterior border of the sternocleidomastoid muscle clearly located and an incision (Fig. 4) made along its edge extending from its sternal attachment to the mastoid tip.[3] Bleeding vessels should, of course, be compressed at all stages of the operation so that a clear view of the operative field may always be obtained. The skin and other tissues should be kept stretched apart by blunt retractors. After the skin and superficial fascia have been completely severed the platysma myoides muscle with its oblique fibers can be plainly seen and should be fully exposed and incised along the anterior border of the sternocleidomastoid muscle, which can now be distinctly seen, and which should be fully uncovered by holding back the overlying tissues with retractors (Fig. 5).

The external jugular vein runs down the neck in a line drawn from the angle of the lower maxilla to about the middle of the clavicle and lies between the platysma and the sternocleidomastoid muscle. It crosses the latter muscle at an oblique angle, but does not cross the anterior border of the muscle except at its upper third at the angle of the jaw, where it may be seen, twice ligated, cut between the ligatures and gently retracted from the operative field. If ligation of the internal jugular were the only object to be attained it would not be necessary to touch the external jugular, for the ligature would be applied much lower down, near the cricoid cartilage, but as the intention usually is to excise practically the entire vein, it may be advisable to get the external jugular out of the way. It should be preserved if possible, however, as it performs an important part in caring for the venous blood after the internal jugular is removed.

Retractors should now be placed around the anterior border of the sternocleidomastoid muscle, and the muscle pulled back in order to expose to view the carotid, jugular, etc. This object may be facilitated by tearing and pulling the tissues apart by blunt instruments or the fingers, as care should be exercised that vessels and nerves are, as far as possible, uninjured, a thing difficult to prevent if knives and scissors are employed. The exposure should be made throughout the entire length of the sternocleido muscle from its mastoid to its sternal attachments, for it will be the object of the operation to remove practically all of the vein, and it should, therefore, be completely exposed (Fig. 6).

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[3] Crile and others sometimes cut down on the sternocleidomastoid muscle and expose the vein, by separating the muscular fibers.

The deep fascia of the neck will be seen on retracting the sternocleido muscle, and on opening this and throwing it back the common sheath of the jugular, carotid and pneumogastric nerve may be seen. The utmost care should, of course, be taken to avoid wounding these structures, and as far as possible all sharp instruments should be eschewed in performing the delicate dissection. The internal jugular vein, the common carotid artery and the pneumogastric nerve are all encased in a common sheath. At the superior carotid triangle (the usual center of operative procedures) or at a point about midway between the angle of the jaw and the clavicle, the vein lies to the outer side of the artery, with the nerve between and posterior to both (Fig. 8), resembling, as Deaver says, the ramrod to an old-fashioned double-barreled shotgun.

ANATOMIC RELATIONS.

It should be remembered that while the relative location of these different anatomic constituents differs slightly in various portions of the neck, the vein always lies to the outside of the group and can be distinguished by its bluish-white color, by the fact that it becomes full and prominent on expiration and almost collapsed on inspiration or by pressing on it in the lower portion of the neck, when it will expand with blood (unless thrombosed) and quickly disclose its identity. The common sheath of the vein, artery and nerve should now be opened and also the septum which separates the vein from the artery and nerve, and the vein carefully dissected out, practically throughout its entire extent. The artery is, of course, easy to identify, both from its color and its pulsations, but the nerve may, unless considerable care is taken, easily be raised and ligated with the vein.

All enlarged and diseased glands should be removed. After the vein has been thoroughly liberated from its surroundings, the lower ligature should be placed as far down in the neck as possible, and the upper one high up, as close to the mastoid process as possible, using either ten-day chromicized catgut, black silk or Pagenstecher linen, preferably the first, as there is practically no danger nowadays in the use of catgut prepared by modern methods. Two ligatures are applied at each extremity by means of an aneurysm needle and the vein cut between each pair of ligatures, being careful to include nothing but the vein in the ligatures.

In order to check the breaking off and floating of small thrombotic particles toward the heart, etc., which may easily occur from the necessary surgical manipulations of the operation, the lowest ligature should be placed around the internal jugular vein as early as possible and as soon as a low exposure can be made, after which the regular steps of the operation may be proceeded with. This lowest ligature can usually be best applied at a point just below the angle of the omohyoid muscle. The tributary veins should be ligated, and they may be enumerated from below upward as follows (Fig. 7):

The middle thyroid in the lower third of the neck (Fig. 7, L); the superior thyroid (Fig. 7, H), a little higher up; the lingual (Fig. 7, F), a little below the lower jaw; and, lastly, the common branch of the temporal and facial (Fig. 7, D) veins. Sometimes these veins are so small as not to require ligating, with the exception of the common branch of the temporal and facial veins, which always requires careful attention. In removing the internal jugular it should always be severed above the posterior belly of the digastric muscle (Fig. 7, A) and above the common trunk of the facial and temporal veins, but the ligation need not extend more than half or three-quarters of an inch higher than this junction, as no more cervical veins enter the jugular above this point.

All of the veins which may require ligation enter the internal jugular (practically) in its anterior portion which if borne in mind will simplify their finding. After all connections are severed the vein may be removed and the parts thoroughly irrigated and cleaned preparatory to closing the wound, remembering that the operation should be performed with all the speed that is compatible with correctness and safety, as such patients should not be kept under anesthesia any longer than is necessary. Sometimes inflammatory adhesions will so firmly unite the vein to its surroundings that its release will necessitate the expenditure of too much time, to say nothing of the danger of rupturing the carotid artery. Under these circumstances the vein and its tributaries should be split open, cleaned, and the neck wound dressed without permanent sutures.

It is not advisable to undertake to irrigate the sinus and what is left of the jugular, either from below upward or the reverse, as infection can easily pass into the inferior petrosal sinus. It sometimes happens that even after the jugular has been resected the upper ligature removed, and the sinus thrombus removed by curettement, there is no return flow of blood from the bulb, which indicates a thrombus of the inferior petrosal sinus. This is an infrequent complication, as is also a similar condition of the superior petrosal sinus, and should be met by following up the sinus and curetting it. The sinus should be cleaned of all possible thrombotic material by the curette after the jugular ligation, but forced flushing should not be attempted.

It is a good plan after the jugular has been resected to remove the upper jugular ligature and give the inferior petrosal sinus an opportunity to send its blood down through the opened jugular vein, in hopes of flushing away thrombotic debris that might be present. The vein should then be resutured, or, as suggested by A. Knapp, "on the day following the operation thrombosis having occurred in the jugular vein, below the bulb, this vein should be slit up, so that we have access to the bulb from both directions, thus preventing a purulent disintegration of the thrombi in the contributory veins and sinuses." Knapp recommends this procedure, especially in the severest cases.

TREATMENT OF WOUND.

The wound should now be drawn together except at its upper and lower extremities, which should be left open for drainage. The lower opening need only be very small, just large enough to admit of a round rubber or cigarette drain, which should extend inside the wound for perhaps two or three inches, and which should be gradually shortened from day to day until it is withdrawn altogether (Fig. 9). The opening in the upper portion of the wound should be larger and should be either packed with iodoform gauze or drained with a round rubber or cigarette drain to be likewise gradually removed until compact healing occurs. Many surgeons do not suture this large neck wound, but draw the parts together with adhesive strips, feeling that the patient should be given every chance for his life by an open wound and free drainage. This, of course, involves an enormous scar but, nevertheless, is justified in bad cases, and possibly always. The jugular operation having been completed, the surgeon should now return to the sinus, on which work had been temporarily suspended.

The compress should be removed from the bulbar end of the sinus and all possible clot removed by means of a curette, after which narrow iodoform gauze should be gently but firmly packed into the interior of the sinus as far as possible. Pressure should, of course, be retained on the torcular end of the sinus and the entire mastoid wound dressed as usual, endeavoring as much as possible to separate the sinus from the mastoid packings. The neck should be abundantly dressed with gauze and cotton and the mastoid, head and neck enveloped in a voluminous and firm bandage, which it is best not to disturb for at least three days. Ordinary dressings at intervals of two or three days should follow until complete healing occurs.

TREATMENT OF SHOCK.

Should symptoms of shock occur during the steps of the sinus or jugular operation relief may be obtained by injections of physiologic salt solution into a vein, the breast or abdominal tissues or the bowels. If an intravenous injection is made, from sixteen to eighteen ounces of the solution should be used at a temperature of about 106° F. If the injection is made into the tissues or bowels from one to two pints should be used at a temperature of about 112° F. At all sinus and jugular operations appliances for making these injections should always be ready for instant use. In case the patient is in a very grave condition it is a wise precaution to give an intravenous infusion of physiologic salt solution before beginning operative procedures. Such infusions are also valuable in cases of general infection to counteract and combat the potentiality of migrating microorganisms.

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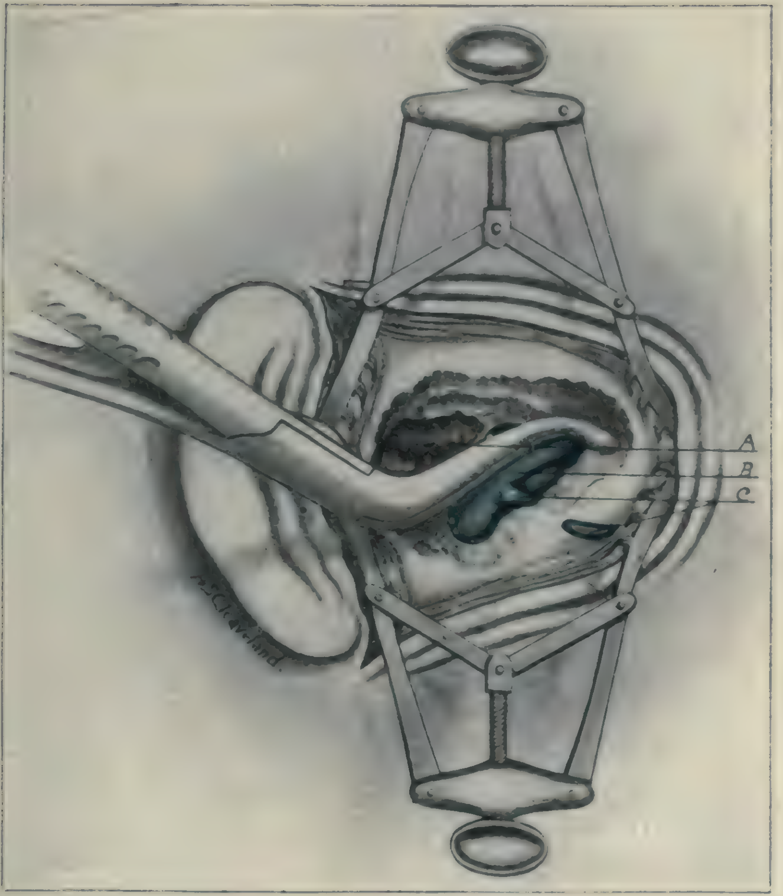

FIG. 1.

Exposing the sinus.
A—Mastoid antrum.
B—Sigmoid sinus.
C—Mastoid emissary vein

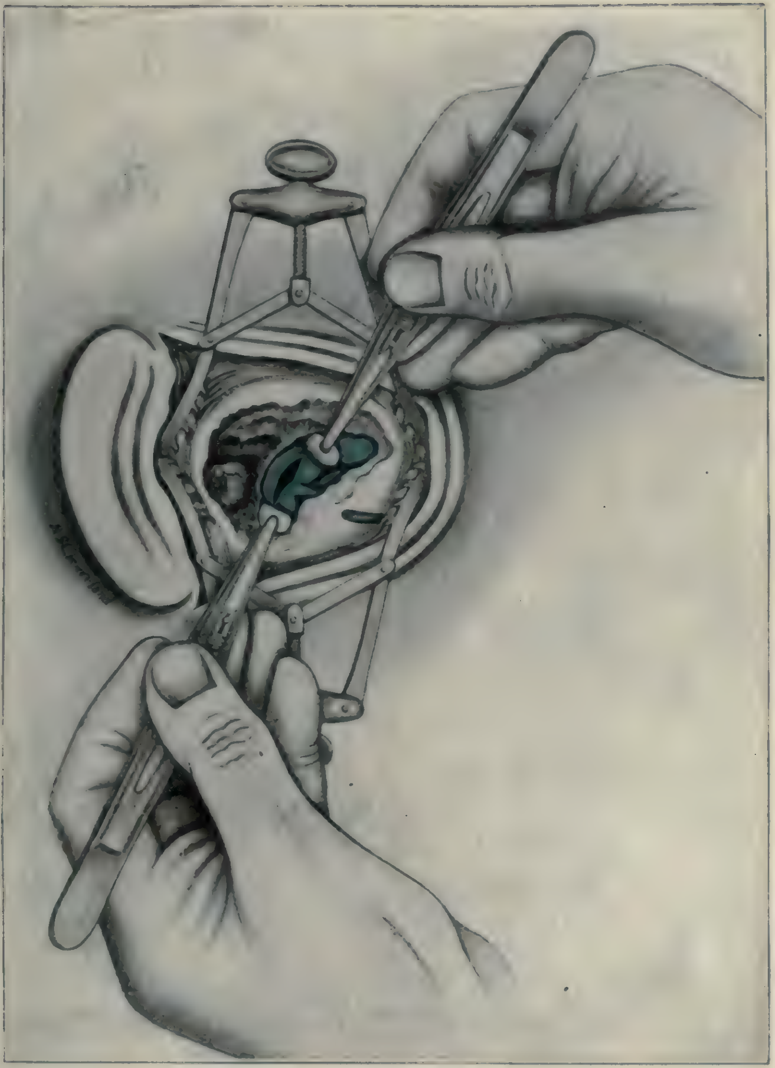

FIG. 2.

Compression of sinus to arrest hemorrhage, and to ascertain the presence of a thombosis.

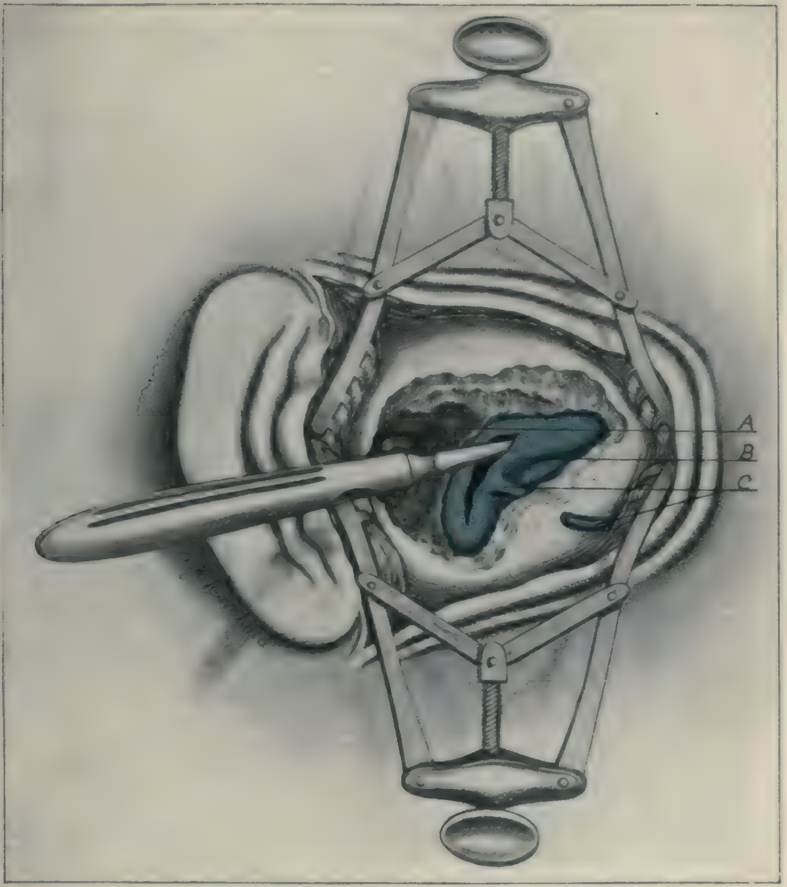

FIG. 3.

Curetting the sinus.

A—Mastoid antrum. B—Sigmoid sinus.
C—Mastoid emissary vein.

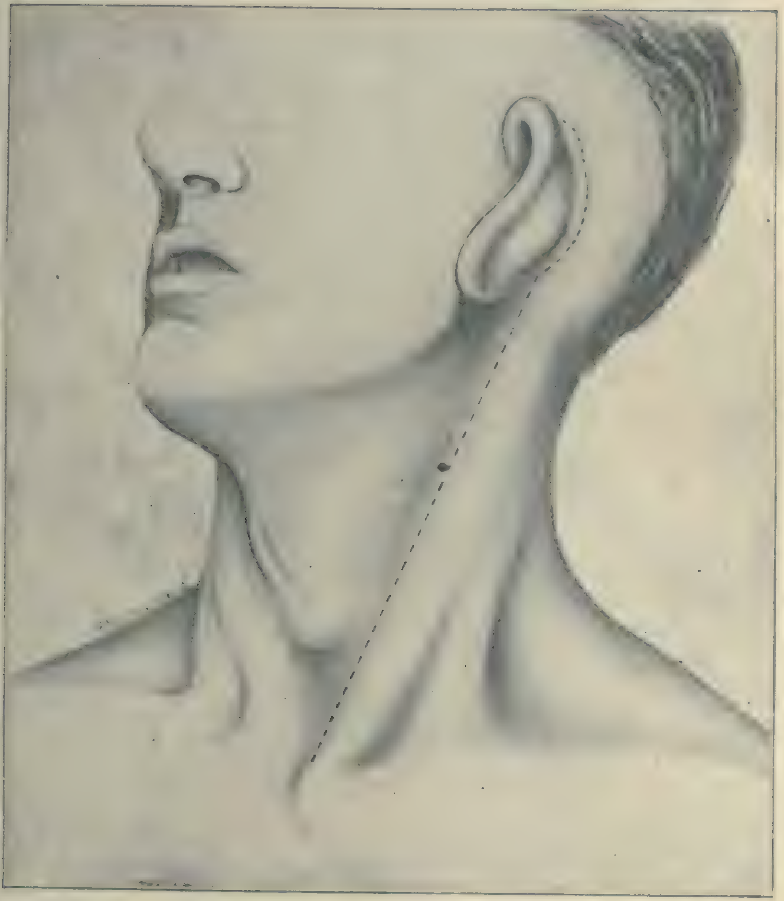

Fig. 4.—Initial incision for exposing internal jugular vein.

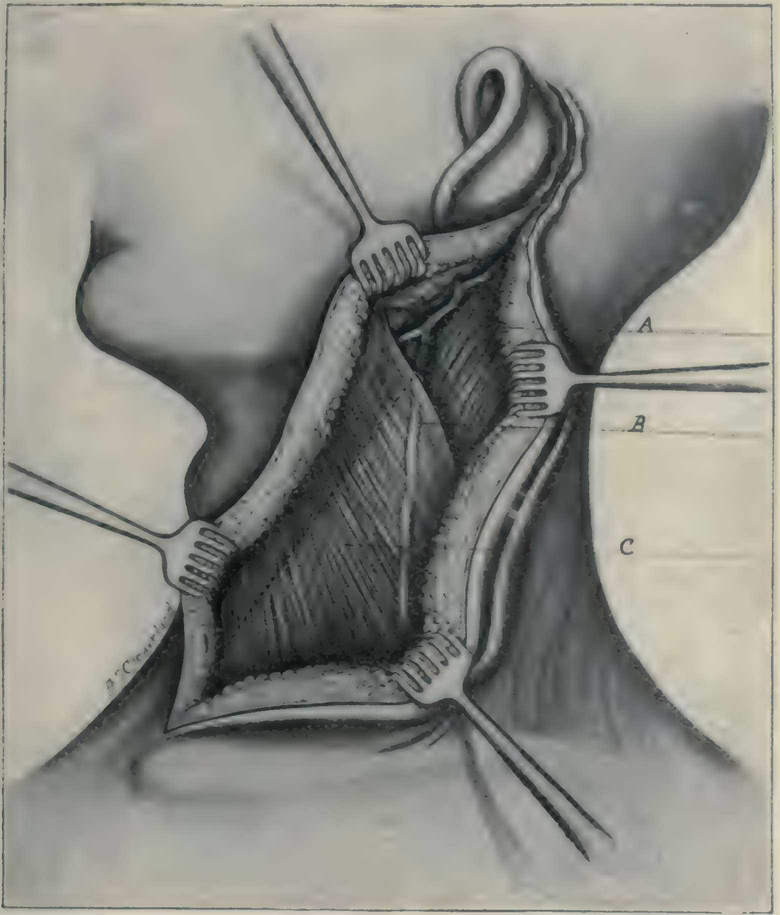

FIG. 5.

Exposure of the platysma myoides muscle and external jugular vein.
A—Sternomastoid muscle. B—External jugular vein.
C—Platysma myoides muscle

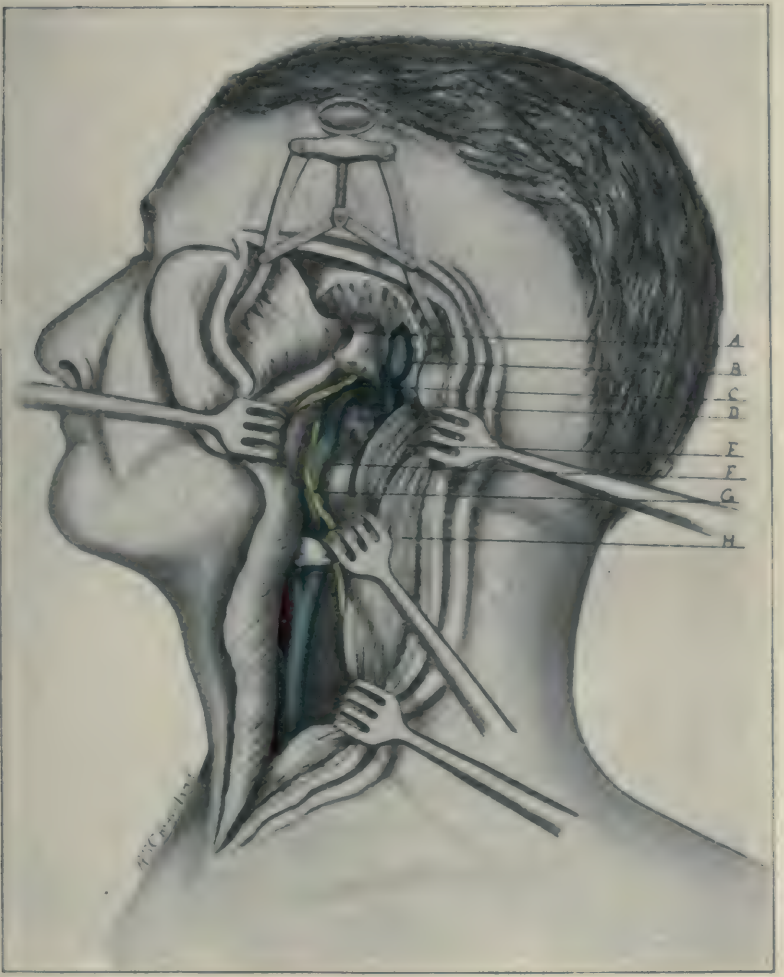

FIG. 6.

Demonstrating the course of the sigmoid sinus, jugular bulb and internal jugular vein.

A—External auditory meatus.
B—Sigmoid sinus.
C—Facial nerve.
D—Jugular bulb.
E—Digastric muscle.
F—Internal jugular vein.
G—Spinal accessory nerve.
H—Sternocleidomastoid muscle.

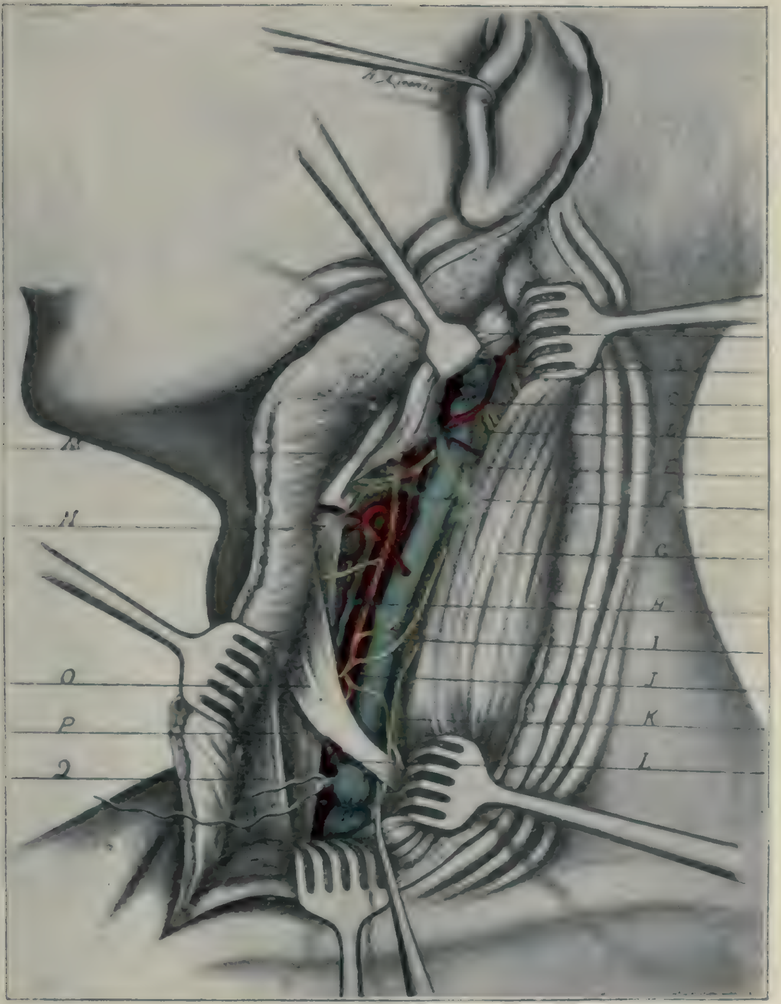

FIG. 7.

Exposure of the internal jugular vein, etc. The vein is about ready to be removed.

A—Posterior belly of digastric muscle.
B—Spinal accessory nerve.
C—Occipital artery,
D—Facial vein.
E—Hypoglossal nerve.
F—Lingual vein.
G—Sternocleidomastoid muscle.
H—Superior thyroid vein.
I—Communicantes hypoglossi nerves.
J—Common carotoid artery.
K—Phrenic nerve.
L—Middle thyroid vein.
M—Stylohyoid muscle.
N—Descendens hypoglossi nerve.
O—Anterior belly of omohyoid bone.
P—Sternothyroid muscle.
Q—Sternohyoid muscle.

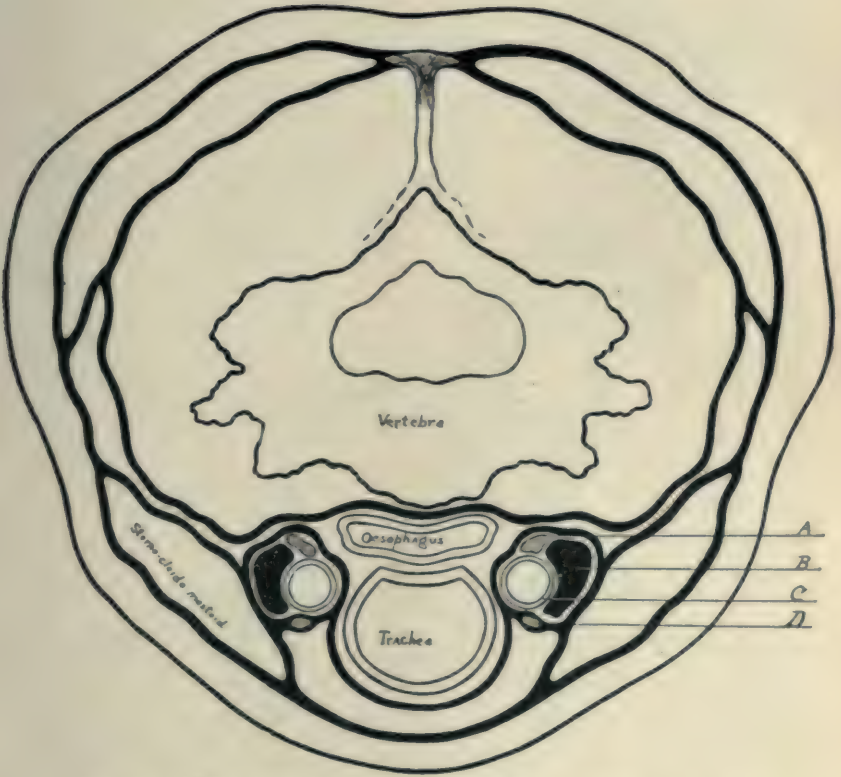

Fig. 8.—Cross section of the neck, after Deaver. A, pneumogastric nerve; B, internal jugular vein; C, common carotid; D, descendens hypoglossi nerve.

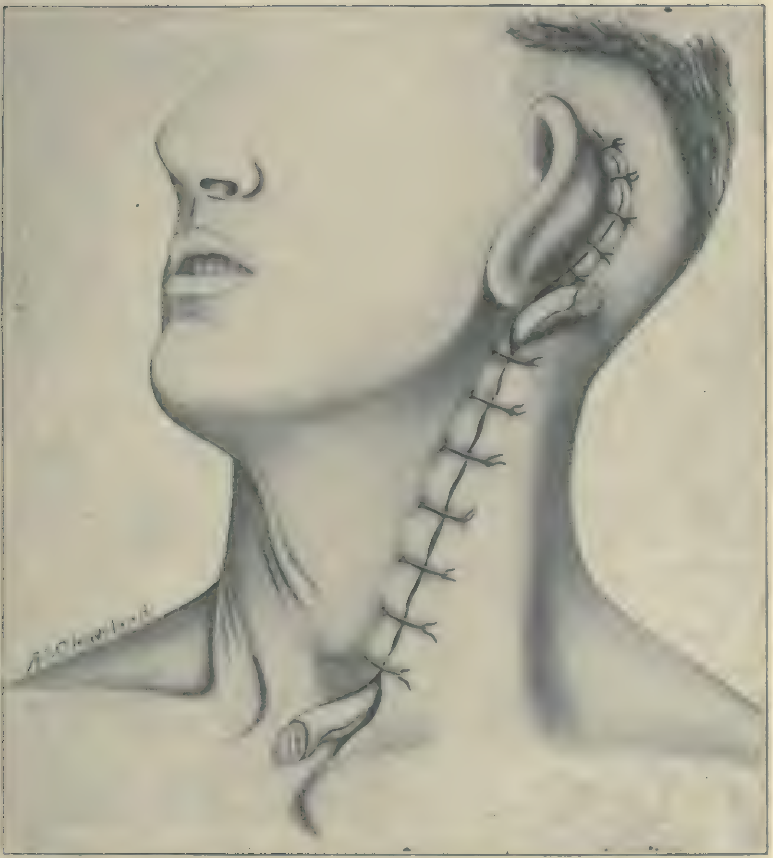

Fig. 9.—Closure of the wound, drainage, etc.

XI.

TREATMENT OF THE INFECTIVE LABYRINTHITIS AFTER FIFTEEN YEARS' EXPERIENCE.*

By Dr. Albert Jansen,

Berlin.

When the title of my paper was forwarded I was unacquainted with last year's transactions of the American Laryngological, Rhinological and Otological Society, and consequently the excellent papers upon this subject, especially that of Dr. Richards, were unknown to me. Had it been otherwise, I should certainly have selected another subject, being fully aware that, from my observations, not many new or original facts can now be presented.

May the fact of my having proposed the labyrinth operation, and of having carried it out for fifteen years, serve as an excuse in your eyes for the choice of this subject.

My observations are based upon upwards of one hundred labyrinth operations. In only two cases was the cochlea alone operated upon. In twenty-three cases the cochlea and vestibule were opened together, and in the remaining cases only the vestibular apparatus. Only five of these cases followed acute middle ear suppuration.

Among those operated upon, 29 died, or 25 per cent, excluding four, in which death was not due to the labyrinth suppuration.

In sixteen cases the operation was performed after purulent meningitis already existed, and death could consequently not be prevented. In three others death ensued undoubtedly from brain complications, like cerebellar abscess, caused by the labyrinth infection.

I propose to arrange my subject synoptically into three or four groups.

The first group which, from its origin and course, is clearly distinctive from the remaining cases, comprises the infectious labyrinth affections caused by lesions of the labyrinth, principally at the oval window.

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*Presented at the fourteenth annual meeting of the American Laryngological, Rhinological and Otological Society, at Pittsburg, Pa., May 28, 29 and 30, 1908.

In this first group of cases we are fortunately able to observe the origin of the disease. Therefore, the symptoms observed assume a decidedly diagnostic importance.

Injuries to the labyrinth are frequently caused by the radical operation. The horizontal semicircular canal is the part most liable to injury; this injury is well known and comparatively frequent.

But of this kind of lesion I shall not speak here. I will only mention that it is generally recognizable at the moment of origin. It seldom leads to operative opening of the labyrinth or to fatal consequences.

In any case it is unnecessary to perform at once an opening of the entire vestibular portion on account of this lesion. Certainly the necessity may arise during the process of healing, but it is evidently exceedingly rare, and I can recollect only one case where the opening of the vestibular apparatus was afterwards necessitated.

We shall here treat of the lesions of the labyrinth, which are much more dangerous in their consequences; namely, the lesions of the fenestra ovalis and the luxation of the stapes.

This part of the middle ear is the one most exposed to lesion from curetting, and, excepting the horizontal canal, the part mostly endangered at the radical operation. Sometimes the luxation, in the case of stapes disease, is very easily produced. Through the accidental removal of the stapes, the vestibule is exposed to serious danger, but the accident is more readily recognized and the danger thereby diminshed.

As a rule, however, it is only a question of luxation of the stapes, of a partial rent of the ligamentum annulare, of an insignificant injury which, in opposition to the injured horizontal semicircular canal, and to the removal of the stapes, we do not notice at the moment it occurs, and which we perceive afterwards only from the symptoms.

That we are dealing with a luxation of the stapes is at first merely a supposition. Experience teaches us, however, that this supposition generally proves correct. The great danger of the stapes luxation lies in the fact that under necrosis it occurs unnoticed and that infection of the vestibule immediately results.

In its consequences, as a rule, in the beginning, such an injury is far more disastrous than the opening of the semicircular canal, while the initial clinical symptoms are usually far less alarming.

Half a day or more elapses before the first signs of labyrinth infection appear, and generally still more time is lost before they are properly appreciated. When the accident occurs, let us say, from curetting in the after-treatment of radical operations, we can make the following observations: Lesion of the labyrinth at once manifests itself in vertigo, disturbances of equilibrium and nystagmus.

It is true that these labyrinthine symptoms sometimes assert themselves only for a moment, coming like a flash of lightning and as quickly vanishing, often so suddenly that the nystagmus is no longer demonstrable.

Directly after the lesion, owing to the appearance of vertigo, the head jerks backwards or forwards, away from or towards the doctor. Sometimes both the head and the upper part of the body make this jerk. Not infrequently there is nausea or vomiting. For a few moments the patient is seized with violent vertigo; he is afraid of falling off the chair, everything seems to be turning round him or with him. On rising, he seems unsteady on his feet, totters or falls.

Occasionally a subjective sensitiveness of the auditory nerve is present in the form of ringing or buzzing. On examining quickly for nystagmus, it is usually found to be present, sometimes with quick, short oscillating movements rather than with slow and long movements, and always strongest when the eyes are directed to the healthy side, or only in this direction. Vertigo appears sometimes only for a moment; sometimes it persists for several minutes, sometimes for a few hours.

Frequently, on account of the overpowering influence of pain, the patient is not conscious of vertigo when it is of short duration, and does not complain of it. The physician mistakes these jerky movements caused by vertigo for a reaction from pain. Even after a few minutes the patient can often rise from his seat and move about again quite well. The nystagmus often disappears, too, and only returns as soon as the infection is established, but continues in case of labyrinthine hemorrhage. It is very easy to overlook the lesion with such feeble labyrinth symptoms. And now, when we have an out-patient to treat, we are confronted by a disastrous chain of circumstances.

The dressing of the wound is done in the usual way, and as the scraping out is often followed by hemorrhage, the packing is done tighter than usual, for hemostatic purposes. This is another unfavorable moment, and facilitates the infection of the vestibule. The patient returns home; soon he feels worse, and goes to bed. The following day he does not feel able to go out; he stays in bed, the dressing remains. The infection progresses and the case ends fatally.

A much more favorable view may be taken of the patient's fate if the damage is perceived at once, or if the patient is undergoing clinical treatment, and consequently, an early ascertainment of the labyrinthine lesion is possible. The damage being at once perceived, a great deal may be done to render the complications less destructive in their course.

One must avoid any manipulation which is not absolutely necessary; powder with iodoform, push in loosely a piece of gauze for absorption of secretions, and attend to the constant discharge of secretion by frequent change of dressings. With extreme care and absolute rest in bed, labyrinth infection is not infrequently prevented.

My cases of labyrinth injuries are 19 in number:

1. Accidental extraction of the stapes	1
2. Luxation of the stapes (the diseased one) during dressing	1
3. Luxation of the stapes (the diseased one) during curetting	7
4. Luxation of the stapes (the diseased one) during the radical operation	6
5. Fissure through the horizontal canal	1
6. Lesions by galvano-cauterization*	3
	19
7. Infection by the curetting of a labyrinth previously exposed by operation	4

Spontaneous cure occurred in two cases; in the one, after scraping out, in the other one after accidental extraction of the stapes. In the latter case the symptoms from the labyrinth lasted several days; they soon diminished and at last disappeared entirely with good hearing.

The second was affected with severe labyrinthine symptoms after scraping out, and subsequently for some weeks with severe symptoms of meningitis. At examination a good many years later the ear was deaf without caloric reaction. There was no disturbance of equilibrium.

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*These cases showed a slow course.

Thirteen were operated upon with four fatalities, 37.7 per cent.

Life was saved:

1. In 3 cases with the complication of serous meningitis.
2. On account of the severe extension of the labyrinth disease in two cases.

It is interesting to know how long after the infection death occurred.

I. In the non-operated cases:
  - In 2 cases after 7 days.
  - In 1 case after 11 days.
  - In 1 case after 18 days (empyema of the saccus endolymphaticus).
  - In 1 case after 37 days (galvano-cauterization).

II. In the cases operated upon:
  - In 2 cases after 6 days.
  - In 1 case after 9 days.
  - In 1 case after 11 days.

III. In those cases which were curetted after opening the vestibule:
  - In 1 case after 5 days.
  - In 1 case after 11 days.
  - In 1 case after 27 days.

In half of all cases death occurred in from 5 to 8 days.

Further, it is interesting to note on which day after infection the operation was performed.

I. In the cases resulting in recovery:
  - 1 after 24 hours.
  - 1 after 27 hours.
  - 1 on the fourth day.
  - 1 on the sixth day.
  - 1 on the eighth day.

In two cases two months after operation and in the cases arising from galvano-cauterization:
  - 1 case the thirteenth day after infection.
  - 1 case the fifteenth day after infection.

II. In the cases resulting in death:
  - The second day after lesion, 4 days before death.
  - The second day after lesion, 4 days before death.

The third day after lesion, 6 days before death.
The fifth day after lesion, 6 days before death.
The tenth day after lesion, 1 day before death.

We see that in two cases the operation was performed too late, even on the second day after lesion; also in another case, on the third day after the lesion, whilst still another case resulted in recovery, though the operation was performed four days after lesion, and recovery has even taken place, eight days after lesion, as well as two months after. The longest period intervening between the operation and death was six days.

The question remaining for discussion is, whether in these cases of injury to the labyrinth death was accelerated or occasioned by the operation.

In the cases presented early for postmortem examination or for operation, the tissues within the labyrinth always showed a hemorrhagic appearance.

The membranous labyrinth was deeply stained with blood, sometimes blackish red, always swollen, sometimes considerably so; the earlier seen the more hemorrhagic it was. As a rule, the process was somewhat more advanced in the vestibule, sometimes when older it showed a greenish discoloration, occasionally also of the bone. The contents of the membranous canals were mostly light red, occasionally dark red.

In one case, indeed, there was after 27 hours, a chocolate-colored mass in the horizontal semicircular canal, the vestibule showing a fresher red. In the early cases no pus whatever was found at operation or postmortem. In one case of postmortem examination after 18 days, the vestibule and cochlea were found filled with pus, also the much dilated saccus endolymphaticus. In cases that came under operation or postmortem later, great destruction was evident in the labyrinth.

These cases, if not operated upon, would probably have met the same fatal end. There is nearly the same period between death and lesion in the operated and non-operated cases. Thus we cannot conclude that the death was accelerated or occasioned through the labyrinth operation.

Some of these lesions were rapidly followed by a very infective purulent meningitis. One patient showed the characteristic symptoms of meningitis two days after the injury to the labyrinth.

As only two out of the 19 cases resulted in spontaneous cure, that is, 10.5 per cent, and even 1 of 17 with luxation, that is, 5.5 per cent, we come to the conclusion that in the case of a determined labyrinthine infection it is better to open the infected part than to wait long for the labyrinth symptoms to increase or diminish.

But how are we to recognize labyrinthine infection? By the rapid increase of the labyrinthine symptoms, often in connection with the signs of inflammation: headache, coated tongue, fever, reactive inflammation of the meninges.

According to pathologic facts the question may arise whether there exists:

1 The purely traumatic injury only.

2. Simple irritation with or without injury, with or without hemorrhage (labyrinthitis serosa hemorrhagica).

3. Infectious labyrinthitis.

In the first two conditions there is a good chance of spontaneous cure, while in the third there is a great tendency to go on to fatal ending. In the first group, inflammation is wanting; in the second group, inflammation is slight; in the third group, inflammation and infection are severe.

In most cases we have an injury of the vestibular apparatus, and in consequence symptoms of vestibular irritation; that is, first of all, dizziness, disturbances of equilibrium, nystagmus. If these symptoms are produced, for instance, by pressure upon the loosened or luxated stapes, the loosening of the dressing is sufficient to remove them. But if they continue, and even increase, they are proof of a severe and growing disturbance in the labyrinth that cannot be explained by the luxation of the stapes alone.

Sometimes the hemorrhage gives the explanation, but that is seldom the case. The hemorrhage may be caused by injury as well as by infection. If now pulse and temperature increase equally, the situation becomes clearer, but at the same time more dangerous. The higher temperature is hardly produced from the infected vestibule or labyrinth, but in most cases (together with the pulse augmentation) by the reaction of the arachnoides. As soon as the temperature increases, be it perhaps only to 38 degrees or so, the indication to open the labyrinth is absolutely clear; but it would be wrong, in case of no fever, to neglect the other symptoms. We have seen that in one case the malady continued for 8 or 10 days without fever, the latter only setting in 12 hours before death.

In cases without fever also it seems to me more desirable to perform the operation than to leave it, as soon as the symptoms of labyrinthine infection are marked, persistent and on the increase, because it is much more probable that the malady will develop into an extensive labyrinthine affection, which would demand an operation later, perhaps under less favorable circumstances. Even an exceptional spontaneous cure cannot turn me from this opinion.

How are the labyrinthine symptoms represented? There are nystagmus, vertigo, disturbed equilibrium, as symptoms of vestibular affection; the loss of hearing is the sign of the cochlear affection. In cases of lesions of the oval window all these symptoms of labyrinthine disease must be searched for in the early stage; they are not so evident as to obtrude themselves upon our notice. If the patient remains in bed after the operation these disturbances are not apparent. Nystagmus is slight but distinct. On looking straight in front the eye seems to rest quietly. On examining sharply with a bright light we observe, when the eyes are directed straight in front, a fine, slight, rapid rotary nystagmus, oscillating towards the healthy side. As soon as the eye passes the middle line towards the healthy side, the twitchings increase, moving in long jerks, and being either rotary or horizontal. The nearer the end-point the more extended the twitchings. They are often now so numerous that they cannot be counted, mostly remaining just about countable. In five seconds one may count five, ten or twelve oscillations, which are not regular and which sometimes cease.

In slight cases the nystagmus ceases if the eyes are directed toward the affected side. In severe cases it continues; and in other less severe cases it reappears, but then oscillating toward the affected side and more slowly. The growing intensity of the twitchings is a symptom of the progress of the inflammation in the vestibular apparatus.

Lying in bed, dizziness is scarcely or not at all observed. The patient lies with closed eyes, his head pressed into the cushions, without moving. He avoids movements, which might produce dizziness, and so the patient, not being aware of the sense of dizziness, cannot complain of it. As soon as he is made to turn upon the injured side or to sit up, he will feel dizziness. Should the patient be requested to get up, vertigo would be more pronounced. When the patient is in bed disturbance of the equilibrium does not appear. If free sitting up in bed is impossible without effort and wavering, the disease must be a very severe one. In the early stage the patient is often able to walk without any unsteadiness. The patient himself may not have the slightest sense of disturbed equilibrium, not even when walking with closed eyes. If asked to place one foot before the other, there comes a slight wavering, sometimes only when the eyes are closed. With this experiment of standing with one foot before the other and the eyes closed, one is able to prove any degree of disturbance of equilibrium which otherwise is not observed.

The caloric reaction, which serves us so well in the diagnosis of labyrinthine disease and gives us very exact knowledge of the degree of sensitiveness in the vestibularis, is really of no use here. In these cases where we must presume that there is an artificial passage, made by injury, leading to the labyrinth, we have to avoid syringing as long as we may hope to get a spontaneous cure. But putting in cotton with cold or hot water instead of syringing does not give reliable results. Moreover, we are observing the labyrinthine inflammation from its beginning, when there is no weakness of the vestibularis. The inflammation may rapidly progress toward the brain before the loss of the vestibular function. We should not forget to enumerate here headache and coated tongue as signs of an inflammatory process in the vestibule, although they may be produced by meningitis as well as fever. We do not doubt that in all these cases of labyrinthine injury in the early stage the vestibularis will very well react to cold and heat.

In this early stage we are not often able to diagnosticate an infective meningitis. The symptoms are still deficient. By the lumbar puncture we may in the earliest stage find a minimal augmentation of pressure, which, however, may quickly rise higher; a few times we found diplococci in clear fluid, once even after 24 hours; a little later we find more leucocytes with it, and a turbid fluid. Among my cases, however, there are some of purulent meningitis without any considerable pressure augmentation, with no bacteria in the lumbar fluid, and even a clear fluid. That is why the negative result of the lumbar puncture can scarcely be used as a contraindication against the operation. The operation ought to be done as soon as possible, in order to prevent the development of an infective purulent meningitis.

The following clinical picture is typical for a severe and progressive infection of the labyrinth:

The patient lies apathetically in bed, avoids moving his head or even raising himself in bed, refuses food and drink because of the inclination to vomit. He lies quietly with closed eyes, mostly on the healthy side; from time to time vomiting occurs, especially after taking nourishment or after moving. The tongue begins to get coated. Slight headache appears. On attempting an upright posture or on turning around he becomes dizzy. Sitting in bed is difficult or impossible. He gives the impression of being very ill. Nystagmus is manifest in the above-mentioned way, at first slight twitchings, increasing to quicker, more lengthened and irregular twitchings, rotary and horizontal oscillations, ten to twenty and more in five seconds, scarcely countable. They are very strong in the end position to the healthy side, continue looking to the affected side.

It is only when the patient gets up that disturbances of equilibrium are observed; at first by standing with feet placed one before the other, in severe cases with open eyes, or standing with feet together and eyes closed. At this stage I consider an operation indicated, although there may be no increase in temperature. In case of the slightest increase in temperature, if this is not due to the general wound conditions, the operation is imperative and must be performed immediately.

We have seen that in the opening of the vestibule the operation was performed too late even two days after the lesion. If the infection has penetrated from the vestibule to the arachnoid it is uncertain whether we shall be able to check it. Unfortunately, we have often a streptococcic infection. On the other hand, if the infection has not yet penetrated the nerve canals, we may be able to arrest the process by a wide opening of the vestibule.

Symptoms of the cochlea are inconstant. Sometimes humming or ringing is experienced, frequently nothing.

The arachnoid membrane is exceedingly early affected by the inflammatory processes of the vestibule. In two cases lumbar puncture proved the existence of pneumococci in the fluid 24 hours and 27 hours after infection. The pressure had risen only very slightly to 150 mm. In this early stage leucocytes are not yet present. In another case lumbar puncture showed a pressure of 220-300 mm.; it was transparent, free from bacteria and leucocytes. Once it was found transparent after 48 hours, and had only a pressure rising to 150 mm. In one case the liquid was clear even after four days, with a pressure rising to 290 mm. In a fifth patient, however, the cerebrospinal fluid was turbid, but free from bacteria, with a pressure of 220 mm.

Just in the first few days when we are looking for symptoms to fix the diagnosis and to decide the necessity for operation, the lumbar puncture seems to give us rather unreliable results, though in spite of this it ought to be done.

Fortunately, not all cases end so tragically, in which, directly after the operation, we notice those disquieting symptoms indicating labyrinthine affection. In my cases I have been able to include twelve with important labyrinthine symptoms arising directly after the operation or a few days later, dizziness, vomiting, nystagmus, disturbed equilibrium.

All these cases were cured promptly.

Moreover, in a number of cases—eleven or more—labyrinthine symptoms appeared during the first week after the operation.

With these patients the symptoms of labyrinthine irritation were of long duration, for weeks and even months.

Two possibilities are here presented. Either in all cases, or in a part of them, there is a luxation of the stapes or an infection of the vestibule induced by the injured cochlea, or there is a latent labyrinthitis, aggravated and made manifest by the operation or by the irritation accompanying the wound healing.

At the same time these cases prove that symptoms of labyrinthine disease do not give us a right immediately to operate in every case, or to oblige us to open the vestibular portion, before satisfying ourselves of the infectious and progressive character of the complication.

My numerous observations of the labyrinthine lesions have taught me that the greatest danger is imminent from the infectious disease of the vestibular portion and essentially from the vestibule itself. After having laid bare the vestibule we may generally consider the danger obviated. We usually combine the exposure of the vestibule with that of the ampullae. But we do not pay any attention to the semicircular canals, from which we fear no danger. There are no free pathways by which the infection from the semicircular canals could get into the inner portion of the cranium. The smaller a canal is the sooner it is cured of inflammatory processes.

Although there exist some free channels leading from the cochlea and capable of conveying infection into the inner portion of the cranium, we have to deal with narrow canals similar to those of the semicircular canals, and the danger becomes greater only when, by bony destruction, the narrow canals form a larger cavity with or without sequestra.

This seldom or never happens in cases of a rapid course. The infection arises in the vestibule, the disease extends into the vestibular apparatus, the symptoms are those of vestibular irritation, and if we interfere as soon as the welfare of the patient demands, a severe, infectious process can scarcely have had time to develop in the cochlea.

These theoretical considerations agree with the experience we have had in such cases, and the results which I have already stated.

The circumstances change when the infection is produced in the cochlea, or when the cochlear irritations have a share in the form of the disease known as humming or ringing in the ear, or noises of various kinds, or in processes having a slow course, where the development of the disease took many weeks so that severe affection of the cochlea may have taken place. Then the exposure of the cochlea may be indicated.

In the literature we find numerous proofs of this complication, as in the pages of Hinsberg, Freytag, Friedrich, Heine, Zeroni, Alexander, but I have noticed in these reports that rather too often the labyrinth infection is interpreted as a latent labyrinthitis, aggravated by the radical operation.

Operations on the Labyrinth Resulting in Recovery.—I come now to the second group. There are 62 patients. The vestibule was opened 63 times, the cochlea 11 times; in one case the cochlea alone was opened.

Excepting two cases the disease was always chronic middle ear suppuration which had caused labyrinth infection. In the two acute cases a deep extradural abscess penetrated into the labyrinth at the posterior petrous plane.

Occasionally the affection was latent. The diagnosis was made either before operation, as was generally the case, or during the operation, through the evidence of fistulae in the labyrinth wall or in the oval window, which was then usually found to be open. Occasionally the labyrinthine disease appears quite spontaneously during treatment, a long time after the mastoid operation.

In the cases which, before the operation, had shown severe labyrinth symptoms, these were, as a rule, greatly relieved after operation; often, on awakening from the anesthetic, the patients felt free from vertigo and much easier in the head; they could sit up in bed and move much better.

Inversely, those cases which presented slight labyrinthine symptoms frequently exhibited after the operation either slight or much stronger symptoms of nystagmus and vertigo for a short time.

In cases which, before operation, were free from vertigo and labyrinth symptoms, these latter occurred after the operation, now and then in a highly distressing degree.

Moreover, these appearances have been frequently met with by other authors. With striking frequency we read in the history of patients of the great increase in labyrinthine symptoms after the opening of the labyrinth. These appearances were manifest very strongly in one case immediately following the radical operation, when the fenestra ovalis was found open; there was a strong suspicion of traumatic affection, recently caused or increased by probing. The condition found at the operation, however, contraindicated this, since the membranous labyrinth was glazed, swollen, not hemorrhagic.

In some few cases the labyrinth operation produced no changes whatever in the health of the patient; once I even found a child sitting up in bed and playing four or five hours after operation.

In one case violent vomiting occurred at the moment of opening the horizontal semicircular canal, and still another case experienced the sensation of being suspended in the air at the moment of striking off the lower vertical semicircular canal, where the vestibule was opened. The operation was performed without an anesthetic.

In one case a discharge of cerebrospinal fluid came from the vestibule. On the other hand, we never observed any fluid discharge among this series of cases upon the opening of the cochlea; quite in contrast to the cases which ended fatally.

As complications we sometimes find sinus thrombosis without any connection with the labyrinth affection; once, cerebellar abscess twice, purulent meningitis; in a number of cases, serous meningitis.

Serous meningitis is a common complication in infective labyrinth disease. It is principally localized in the posterior cranial fossa, while the middle and anterior fossae are free from it. In the puncture of the dura we found cerebrospinal fluid in great quantities only in the cerebellum.

Several times I could find proofs of an interesting affection of the saccus endolymphaticus.

In several cases I found a circumscribed livid discoloration, about the size of a five-cent piece, on the cerebellar dura, from which the surrounding dura stood out rather sharply, owing to deficiency in livid discoloration and to its less moistened appearance. This area was close to the descending crus of the sigmoid sinus. I have often observed this phenomenon. There is no doubt, in my opinion, that it is a sign of an inflammatory process of the saccus endolymphaticus. The appearance depicted by this circumscribed livid discoloration in the vicinity of the sinus is a very characteristic one, and since recognizing this phenomenon and searching for it I have come across it repeatedly. It is a serous inflammation of the saccus which is in some cases productive of purulent inflammation.

When the radical operation has been performed, the closest inspection of the labyrinthine capsule is necessary.

In 1896 I reported 196 cases of defects of the semicircular canal, which, with few exceptions, were located on the horizontal semicircular canal. Since then I have seen 350 cases of infective labyrinthine disease, of which again by far the majority were fistulae of the horizontal semicircular canal.

Although fistula of the horizontal semicircular canal is much the commonest, fistula in the fenestra ovalis is by far the most dangerous. Exploration of the fenestra ovalis has only been systematically carried out by me of late years. Among the 62 cases resulting in recovery, the fenestra ovalis was found empty in 20 cases, and the horizontal semicircular canal was mentioned as intact in 10 cases; in several cases with transparent lumen.

The presence of fistula in the horizontal semicircular canal is doubtless the chief pathway for the conveyance of infection to the labyrinth; but certainly not infrequently it must also be regarded as an escape from within.

When the invasion of the labyrinth occurs through the horizontal semicircular canal, there is present in many cases a circumscribed labyrinthine disease, the affection being limited to the semicircular canal.

It is by no means always easy to arrive at a correct conclusion respecting the condition of the fenestra ovalis. The fenestra ovalis must be quite exposed for examination which must be conducted with great precision and delicacy, lest we ourselves cause a lesion in or at the window. We must also bear in mind that infection of the vestibule, if the vestibular wall is intact, may arise from the cochlea, which may be infected by an inconspicuous fistula in the promontory, or again, through a diseased fenestra rotunda.

It is now important to decide whether we are dealing with a circumscribed disease of the semicircular canal, with a diffuse disease of the vestibular apparatus, or with a disease of the entire labyrinth.

Unquestionable cases have come under my notice in which the entire vestibular portion was attacked, but where the inflammation was restricted, for instance, to the horizontal semicircular canal. The entire labyrinth can be attacked by a non-infectious inflammation.

The part attacked is, without doubt, either impaired in its functions or completely paralyzed. The cochlea would appear to become paralyzed in its functions more quickly than the vestibularis. The cochlearis is the more highly organized nerve, and the higher the organization, the more delicate it is, and consequently, the greater the liability to decay and loss of function.

The following kinds of labyrinth affection may occur:

I. A serous, non-infectious inflammation of the entire labyrinth, or of separate parts of it, produced by the adjacent middle ear disease, with or without defect in the labyrinth capsule.

II. An infectious disease of separate parts of the labyrinth: (1) the cochlea: (2) the vestibule; (3) the semicircular canal. There may be:

(a) A serous hemorrhagic infection.
(b) A purulent hemorrhagic infection.
(c) A purulent infection.
(d) Infection with necrosis.

The partially infective inflammation may be associated with a diffuse non-infective reaction of the entire labyrinth.

In every case of inflammation of the cochlea the function quickly ceases. If there is a serous affection or a quite slight infectious one, the process may result in recovery with a complete or, more probably, a partial restoration of function.

In a serous or slightly infective disease of the vestibular apparatus, the functional powers appear able to exist for a longer time. Here recovery may also be expected with complete restoration of the function. If the patient's hearing is fairly good, we have, most probably, a circumscribed disease of the semicircular canal.

More important and significant for the diagnosis are the phenomena of vestibular disease, signs of irritation or paralysis of the nerve. In the manifest cases the diagnosis may often be found at first sight.

Barany's method of caloric reaction most promptly acquaints us with the condition of the vestibularis and its functional power. We herewith test its sensitiveness by the application of cold and heat, by temperatures above or below blood heat. We use cold water of 12 degrees to 15 degrees C., and hot water (50 degrees). With susceptible nerves slight differences suffice, with less susceptible nerves great differences in temperature must be applied. The results so obtained are especially valuable in those cases where the labyrinth symptoms have already disappeared, where the labyrinth disease is latent, or where it has resulted in spontaneous cure with paralysis of the nerve.

In syringing with cold and hot water, characteristic nystagmus and disturbed equilibrium combined with vertigo are produced. Nystagmus appears on the application of cold exactly as in the case of labyrinthine suppuration, on the opposite side, and, vice versa, on the application of heat, it appears on the same side, its intensity depending upon the temperature or the pathologic condition of the nerve. We observe here all degrees of nystagmic movements from quite slight, scarcely perceptible, twitchings, to rapid, wide, wild and irregular rolling of the eyes. Similar to this are the disturbances of equilibrium, which sometimes do not exist at all, sometimes only appear in slight degrees, and sometimes in so high a degree that the patient has no longer any control over himself. He is able neither to sit upright nor to stand, and in these severe cases the vertigo produced by rotary movement is extremely pronounced. Everything seems to be turning round him and he himself collapses and falls. In slight degrees vertigo is sometimes completely absent.

The appearances produced by caloric reaction are perfectly in keeping with the phenomena of manifest labyrinthine disease.

Though it was known long ago that syringing with cold water produced vertigo, great merit is due to Barany for having followed up these symptoms most thoroughly, and for having constructed a method of practical use with the patient, which in latent cases of labyrinth disease enables us to arrive at a prompt conclusion of the condition of the vestibularis; but not of the kind of pathologic alterations.

If no reaction occurs, paralysis of the vestibularis is proved. In most cases a purulent or infectious process in the vestibular apparatus will be the cause.

To answer the question concerning indications for operation, the caloric reaction has to be used with precaution, and in those cases of very rapid development of labyrinthine inflammation, which are so very dangerous because of the possibility of a rapid infection of the arachnoid, it is also unsatisfactory. In these cases the caloric reaction has not yet disappeared at a time when an operation would give the best chance. In those cases where the operation allowed me to ascertain the condition of the inner labyrinth, caloric reaction was as follows: Four times negative, twice almost negative, neither vertigo nor nystagmus, but immediately after the test a severe fainting fit occurred, and the spontaneous nystagmus ceased completely. Five times the reaction gave slight results, once with cold water, but no results from hot; four times a slight and three times a very marked reaction.

In those cases with marked reaction the operation gave the following result:

I. Very slight edematous swelling, semicircular canal intact, fenestra ovalis empty and open. In a second case the semicircular canal looked dark in its anterior portion. Fenestra ovalis empty, the membranous labyrinth in the semicircular canal and vestibule succulent and dark-colored. In the cochlea were granulations. In a third case (fistula in the promontory), much hemorrhagic fluid in the horizontal semicircular canal; the contents of the vestibule dark red, hearing much lessened.

In cases with moderately strong reaction the result was as follows: First, the horizontal semicircular canal looked dark through the anterior crus, fenestra ovalis empty, in the vestibule a glazed swelling of grayish-red color.

In cases where there is no reaction whatever the horizontal semicircular canal appears dark in the forepart about the ampullar area. Fenestra ovalis penetrable by the probe. The membranous semicircular canal is yellowish-red, in the area of the ampullae dark brown, and represents here a stout cord. Concerning the contents of the vestibule I could not come to a precise diagnosis.

The above phenomena further show that only the negative result gives a perfect proof; moreover, that we can find with a well-conserved reaction severe inflammatory alterations in the vestibule and semicircular canal, which have often led to fatal meningitis.

The lack of caloric reaction indicates, moreover, a severe affection of the vestibule, but of what kind the changes are, the experiment does not disclose. From the acute hemorrhagic swelling there would usually appear to be an important diminution, but sometimes only a slight one; with chronic glazed swelling, the reaction is often more pronounced. There may be a glazed swelling and even pus in the vestibule, and, notwithstanding, the caloric reaction may still occur in a more or less marked degree; there may be a cicatricial healing process or an ossification of the entire labyrinth, or a complete suppurative decay; there is always the same result—loss of caloric reaction. For the diagnosis of an affection and the functionability of the vestibularis, the experiment is doubtless very valuable, but it discloses nothing of a surgical advantage or of the character of the infection in question.

Do the other labyrinth symptoms reveal anything similar, more important or less so? The remaining symptoms in relation to the vestibularis consist of equilibrium disturbances, spontaneous nystagmus and vertigo, and are very significant, especially in combination with the caloric reaction.

NYSTAGMUS.

As early as in 1893 I described nystagmus as a symptom of labyrinth disease, observed especially when the eyes are directed to the healthy side, and in 1896 I gave a detailed account of a great number of cases showing its frequency, manner of appearance and diagnostic importance. It may often be produced during examination by the application of galvanocautery or catheterization. Since the beginning of 1890 I have made numerous observations on the appearance of nystagmus, vertigo and disturbances of equilibrium after application of galvanocautery. The nystagmus lasts from a few seconds to several minutes. It disappears at the same moment as the vertigo ceases, but a certain weakness or an unsteadiness on the legs remains in some cases for a longer or shorter time.

The reaction is usually very marked. The nystagmus may occur in any form but it is always directed towards the diseased side and is often very intense with extensive, rapid movements. The eyes roll irregularly to and fro, with long oscillations, both rotary and horizontal, so rapid that counting is utterly impossible; the eyes are uncontrollable and can not be fixed on any given object; gradually the eyes get steadier, the movements grow less frequent and smaller, until at last we can count them and at the end they appear only when the eyes are directed to the diseased side; after a few minutes the eye is perfectly quiet again.

This sort of nystagmus is always accompanied by vertigo, which corresponds perfectly to its intensity as well as to the disturbances of equilibrium.

While catheterizing with cold air I have frequently produced nystagmus directed to the side opposite to the healthy one, it having the same effect as syringing with cold water. The sense of cold produces this reaction of the vestibular nerve. This nystagmus is always associated with vertigo. On catheterizing, the irritation is much less than with galvanocautery. Long before Barany's publication, I observed nystagmus following application of the galvanocautery and catheterization with cold air. With the galvanocautery the nystagmus is strong and is directed towards the affected side, while in catheterization with cold air it is directed towards the opposite side.

The appearance of nystagmus and dizziness from pressure upon the auditory canal has long been known; in such a case there is a fistula in the labyrinth wall.

The direction of the nystagmus depends upon the quality or kind of the irritation affecting the vestibular apparatus.

Spontaneous nystagmus is very frequently seen in diseases of vestibular apparatus, generally directed to the healthy side and becoming intense when the eye is turned in the direction of the oscillations. In mild cases it only appears in this direction. It is a sign of greater irritation, if much nystagmus appears in both directions. Slight oscillations of this sort are unimportant.

Nystagmus oscillations always increase if the eyes are turned in the direction of the oscillations, and they disappear by turning the eyes in the opposite direction.

If nystagmus oscillating towards the healthy ear appears also when looking forwards and to the diseased side, it is a proof of great irritation, of severe infection and of rapid progress of the inflammatory process in the vestibular apparatus.

In addition to the nystagmus oscillations we frequently observe a great restlessness of the eyes. After several slight movements to and fro, which are quite easily distinguished from nystagmus, the eyes rest at a fixed position and from this it is sometimes shaken to the right or to the left.

In severe cases of nystagmus there is an inability to fix the eyes on any given object. The eyes do not remain stationary, but deviate slightly to the other side; in very severe cases they turn to the midline and then return with a sudden jerk to the end position.

There is no doubt that the rapid appearance of nystagmus after application of galvanocautery, after catheterizing with cold air, on pressure of the auditory canal, or after syringing with cold and hot water is symptomatic of labyrinth irritation on that side, being the symptom of an irritation of the vestibularis either from treatment or irritation. With different directions of the movements, the oscillations vary according to the gravity of the irritation.

The spontaneous nystagmus produced by ear suppuration has the same cause; at least in the beginning there is an irritation of the vestibularis, somewhat similar to the use of cold water. In many forms of decay of the vestibularis, however, nystagmus may be looked upon as a symptom of paralysis of the nerve. According to the degree and kind of the irritations the oscillations are directed to the healthy or to the diseased side. At present it is not known why suppuration in the vestibule induces nystagmus in the same direction as cold water does. It is singular that with suppuration in the vestibule, nystagmus is usually directed towards the healthy side.

Experience further shows, that the more acute the process in the vestibule is, the quicker will be the movements of nystagmus. A high degree of nystagmus is produced either by a very severe labyrinth inflammation, or great irritation in the labyrinth, for instance sequestra, empyema (under compression), or granulations under pressure. There is also a high degree of dizziness and disturbance of equilibrium. Changes in the labyrinth capsule, sequestra, high outside pressure with fistula may also be the cause. On the other hand, there may be a severe disease of the labyrinth without any nystagmus and strong spontaneous nystagmus alone or with only slight degree of equilibrium disturbances is observed in cases where there is no other sign of severe labyrinth disease.

There must naturally be nerves capable of function to produce this nystagmus. It seems doubtful whether nystagmus can arise from decay of the nerve endings or the nerve trunks.

At all events it is important for us to know that generally, but not always, severe nystagmus is a symptom of a severe affection of the labyrinth.

But what is the state if we have but slight nystagmus or none at all?

In many cases where the vestibularis is completely or almost completely destroyed and consequently its function almost eliminated, we still observe spontaneous nystagmus, usually only in a slight degree, sometimes more, and often entirely absent. From my experience, this kind of nystagmus is always directed to the healthy side and apparently produced by the cerebral center or by the other vestibularis.

From the existence of only slight degrees of nystagmus, we can not conclude that there is only a slight affection of the labyrinth, and it would be quite a mistake to conclude that the vestibule is healthy if there is no nystagmus.

Just in those cases where labyrinth infection is latent, where the nystagmus has subsided in consequence of disease of long duration in the vestibular apparatus, the result of caloric reaction is of great diagnostic importance and renders the affection manifest. The disturbed equilibrium usually corresponds with the degree of nystagmus.

Since we are in possession of the convenient and on the whole trustworthy method of caloric reaction, examination on the revolving chair and turning the patient around several times seem superfluous and are no longer used regularly.

The symptoms obtained from the disturbances of the vestibularis are as already seen numerous and give a sharp clinical picture. The symptoms on the side of the cochlearis are far behind those of the vestibularis in their frequency or in their obtrusiveness. However, there are usually disturbances on the side of the cochlearis, which sometimes are very valuable for the diagnosis.

The symptoms of deafness become characteristic if they develop under our eyes, if we can witness the sudden change of Weber's test, the elevation of the lower tone limit, the lowering of the upper tone limit and the loss of hearing for speech.

The kind and the degree of the equilibrium disturbances give us valuable signs of the intensity of the labyrinth affection, especially for its surgical importance; more so than the lack of caloric reaction, the great value of which consists chiefly in making manifest a latent labyrinth affection. The more severe the labyrinthine symptoms are, the more dangerous is the affection generally, and the more necessary is the opening of the labyrinth.

With the help of the improved diagnostic methods of examination and especially with the assistance of the caloric reaction, we shall generally be able to diagnose the disease of the labyrinth before the operation. This is naturally of great value, for we can begin with the operation well planned in advance.

INDICATION AND PROGNOSIS OF THE LABYRINTH OPERATION.

According to my experience the labyrinth operation is in general not dangerous; the opening of the vestibular apparatus is particularly without danger.

The technic is simple. Danger would only result from penetration of the inner vestibular wall and from injury to the dura in the inner auditory meatus. But this injury is exceedingly easy to avoid when the operation is performed *lege artis*, and when there are present no abnormal conditions such as are seen in the ossification of the labyrinth. Moreover, a highly situated jugular bulb can scarcely signify an appreciable increase of danger to life. Indeed, in consequence of narrow-spaced conditions, a small mastoid process, strong prominence of the lateral sinus, proximity of the cerebellar dura may render the operation difficult to such a high degree that the correct procedure is only with great precaution possible without injury to the adjacent organs.

Moreover, the opening of the cochlea performed *lege artis* appears to be unattended by danger, but having practiced the opening of the cochlea in 23 cases only, while I have opened the vestibule over 100 times, I should not permit myself to make a positive statement upon this question.

It seems certain, however, that in the opening of the cochlea injuries to the adjoining structures, i. e., the dura in the inner auditory canal, occur much more readily, and may be regarded less as technical faults than in the opening of the vestibule.

Further, we have come to the conclusion that infection of the vestibule renders surgical interference necessary much more frequently than infection of the cochlea.

The complications of the deep extradural abscess, of empyema of the saccus endolymphaticus, and of cerebellar abscess, occur only by this localization. The infection invades the vestibular apparatus through the oval window or the horizontal canal. The infectious disease is much more frequent than the cochlear malady, and more extended and more progressive. The cochlear affection when fatal seems to have a slower course. For all these reasons the vestibular apparatus must come first in our consideration of operative treatment.

It will be readily admitted that disease of the entire labyrinth represents a much more serious malady and renders the possibility of extension to the meninges decidedly easier than when only a single portion of the labyrinth is infected. A warning to us are the four cases in which, after a successful vestibular operation, death was produced by meningitis, granulations being scraped out of the vestibule a long time after the operation, or, concurrently with this scraping out, Thiersch's skin transplantation being performed.

These cases demonstrate to us that perfect freedom from danger is not guaranteed even upon the evidence of the safety of the operation; danger to life may still arise from the subsequent treatment. These dangers, however, diminish if they are anticipated, and, accordingly, the Thiersch's skin transplantation, as well as the curetting, must be used with the greatest care, or be entirely avoided.

What indications are to be derived from our observations?

If we appreciate the freedom from danger of the opening of the vestibular portion and consider the dangers resulting from subsequent treatment as avoidable, we should be able to set up the principle, as soon as labyrinth disease has been diagnosed in one of its principal parts, that the operation is admissible in every case in the face of the dangers which may arise to the patient from labyrinthine disease. I may state here that in this time of observation I have seen ten fatal cases among 190 cases of decided labyrinthine disease in which only the middle ear was operated upon. The labyrinth operation was naturally done in the most severe cases. We have indeed acquired evidence, from a great number of positively diagnosed cases of disease, at least of the vestibule portion, in a great number of cases, the labyrinthine disease does not end fatally, nor in a chronic ailment, but in a spontaneous cure.

In my opinion there is not the slightest doubt that in the majority of these cases disease of the entire vestibular portion has been present, even though single or numerous instances of it may be regarded as circumscribed disease of the semicircular canal. We often found, a long time after the cure, negative caloric reaction. We see from this, without doubt, that the labyrinthine disease, in many cases, ends in spontaneous cure, also that it does not always, in fact, incline to progression, but the cure results slowly and labyrinthine symptoms lasts a long time. The labyrinth operation is permissible in every diffuse infectious disease of the entire labyrinth or of one of its two portions, especially of the vestibule.

The operation should not be performed when it is a question of circumscribed disease in the labyrinth wall or in one of the semicircular canals; when the labyrinthine disease is complicated with infectious processes in the posterior cranial fossa, seldom in the middle one; meningitis serosa, cerebellar abscess; caries necrotica of the pyramid. As a rule this applies to the horizontal semicircular canal.

When can circumscribed semicircular canal disease be diagnosticated and consequently labyrinthine operation be omitted?

1. When the labyrinthine symptoms are not present in excess.

2. When the function of the cochlea is not destroyed.

3. When the caloric reaction is marked, or at least well present.

4. When the fenestra ovalis is intact.

5. When acute middle ear suppuration is wanting, because of the tendency to diffuse labyrinthitis.

Carious processes on the outside of the labyrinthine capsule cannot be a reason for performing the opening of the labyrinth.

Operation is strongly indicated:

I. Where serious disease of the entire labyrinth or one of its two parts is present, usually the vestibule being affected.

II. When in stapes luxation, after 24 hours, the symptoms present are increasing nystagmus, equilibrium disturbances and coated tongue, growing worse. If there is fever, it is then most necessary to operate.

When is the serious disease of the labyrinth or of the vestibule alone?

1. In acute middle ear suppuration every vestibular disease is serious and dangerous, and every fistula in the semicircular canal is to be regarded as a sign of a present or imminent affection of the entire vestibular apparatus.

2. (a) Upon acute appearance of severe labyrinthine symptoms.

(b) Especially in severe labyrinthine symptoms, not arising from pressure of cholesteatomata, granulations, pus on the perforated horizontal semicircular canal, the caloric reaction lacking.

3. When, under observation, the caloric reaction is lacking or diminishing, the function of the cochlea diminishes, the vestibular symptoms become gradually worse.

4. Fever, without other causes.

5. In cerebellar complications in the posterior cranial fossa, especially meningitis serosa.

6. Severe labyrinthine disease is further recognizable during the operation by

(a) Appearance of pus, granulations from the fenestra ovalis or another fistula.

(b) More extensive necrosis, especially at the vestibular wall.

Caloric reaction will be lacking or decreased except in 1 and 2, in which it may be reduced. Its absence, however, is not necessary, since it alone does not prove the presence of a severe affection.

I adhere most decidedly to the indication for operating in severe labyrinthine symptoms, not arising from the middle ear. Only when they have existed for a long period and the vestibular reaction is well maintained, as well as the cochlear function, can one expect the result of the radical operation. I lost a few patients because in spite of severe labyrinthine symptoms I could not make up my mind to operate at once.

METHODS OF OPERATION.

In opening the vestibule I have for fifteen years employed two methods, which I made public in various papers in the years 1893-1900. One method accomplishes the exposure of the vestibule, inside the temporal bone, by following along the horizontal semicircular canal; the other one by removing the posterior wall of the petrous bone from the posterior cranial fossa at the upper crus from the lower, and at the posterior crus from the upper canal, as well as at the posterior crus from the horizontal semicircular canal. Injury to two organs must be avoided—to the facialis in front, which may occur in the first method; to the dura behind, which may occur in the second method.

With a high position of the jugular bulb and the fossa jugularis, a lesion of the bulbus is also possible, and if the fossa jugularis stretches very high up into the temporal bone the bulbus, especially when the opening is made from behind, may be very much in the way of the operation, even making it impossible. A lesion of the inner wall of the vestibule, with injury to the dura of the internal auditory canal, is almost impossible, if the operation is properly performed. But caution is necessary, the wall in part being very thin. Injury to the dura in the meatus would have fatal results. I once saw death result. Opening of the internal auditory meatus is quite possible, if one loses the direction and penetrates above or behind the vestibule into the petrous bone, instead of forwards and downwards into the vestibule.

OPENING ALONG THE HORIZONTAL SEMICIRCULAR CANAL—SEMICIRCULAR CANAL OR TYMPANAL METHOD.

I usually begin the operation by removing the upper half of the horizontal semicircular canal, and thus fix upon the anterior crus of the horizontal canal the limit below which one may not go. By operating carefully it is always possible to preserve the lower wall of the anterior crus; then the facial nerve is protected from lesion. At the posterior crus I remove the whole canal and to keep away from the roof of the vestibule. I also remove the bone under this part of the horizontal canal in the direction sloping down to the back, taking the very greatest precautions with regard to the facialis, which lies in front.

The stapes plate reaches with its posterior margin about the lateral boundary of the vestibule and takes up a middle position between horizontal and vertical. By this and by the extension of the ampullar extremities of the horizontal and upper canal, the upper portion of the vestibule leads toward the front.

If I penetrate in this way by the anterior crus in the direcof the canal, and above its lower wall towards the vestibule, and by the posterior crus close to its posterior and lower edge, I have selected a large portion of the lateral wall of the vestibule as the place of attack.

Thus it is easy to open the vestibule either in its posterior lower part, or near the ampulla of the horizontal semicircular canal. According to the pathologic changes, the vestibule is to be opened here or there, in front, above, or at the posterior orifice of the semicircular canal; in cases without fistulae, in the described way, along the horizontal semicircular canal, in the direction of the oval window, parallel with, behind, and a little below the facial bridge. Thus the facialis is surely protected from lesion.

The direction in which we have to seek the anterior wall of the vestibule is indicated not only by the anterior crus of the horizontal semicircular canal, which might lead us astray by its long extended position, but also by the fenestra ovalis, which we can in any case precisely locate by the introduction of a hooked probe. In cases of narrowing of the vestibule by new formation of bone we must expose the front part, at the same time preserving the portion of bone which supports the lower wall of the anterior crus, as a bridge. We penetrate behind and parallel with this bony ridge, inwards, downwards, forwards, in the direction of the fenestra ovalis. If the vestibule is found, the examination with a hooked probe will give a certain opinion as to the size, form and position of the cavity. The lateral wall may easily be felt with the probe, and then neatly removed with a narrow chisel or burr.

If one wishes to obtain information of the contents of the vestibular apparatus, the chisel is to be preferred. To prevent the anterior crus from snapping off, the burr is the better instrument. By using necessary caution and by experience the facialis need not be exposed. It is of the greatest importance to use the chisel as a plane, and to plane off shaving after shaving.

It is of importance to reach the deepest point of the vestibule, to lay bare its floor, to smooth the way to it properly, and to arrange that it may be seen at a glance. By exposing the lower orifice of the inferior semicircular canal we enlarge the cavity of the vestibule downwards, and by exposing the common orifice of the vertical semicircular canal we enlarge it at the back. The burr is eminently adapted to enlarge the vestibule towards the back, either to reach diseased bone lying behind the labyrinth better, or to create below more favorable conditions for wounds, with or without exposure of the dura.

Finally I expose the ampulla from the upper vertical semicircular canal, and then consider the operation completed. Only very rarely do I follow up all the canals, opening them in their full length, starting from the cavity of the wound.

The disease in the canals is usually slight, and after exposure of the orifices into the vestibule, the conditions for healing are specially favorable in them. It is not necessary to expose it further from the fenestra ovalis. Only if the edge of the fenestra ovalis itself is diseased, or the wall of the labyrinth is affected, and an operation on the cochlea becomes necessary, do we consider it expedient to scrape and enlarge the fenestra ovalis with the burr. In general I warn against this, especially against vigorously enlarging the fenestra ovalis towards the back and below, for the bridge which supports the facialis is thus made narrower and necrosis is facilitated. As a rule I employ this semicircular canal method. In fact I consider it the obvious method, in the ordinary forms of labyrinth disease, pointing to the safest way. Any danger to life is out of the question, is impossible.

I should most emphatically reject all attempts to arrive at an opening of the vestibule from the fenestra ovalis only. The widening of the passage is too inconsiderable, and can, in my opinion, not be of sufficient use, even if, by widening downwards to the back, the opening can be enlarged to the floor of the vestibule. The cases which are cured by this method of operation would in all probability have healed quite spontaneously.

ENDOCRANIAL METHOD.

I have much more rarely employed the second method of exposing the vestibular apparatus after removing the posterior wall of the petrous bone and widely opening the posterior, and, when necessary, the middle cranial fossa. I performed this operation for the first time in the year 1895, in the case of a deep extradural abscess which had penetrated into the labyrinth. In the same year I also indicated the circumstances under which I considered it right to employ this method. In Blau's Encyclopedia I have fully described both methods.

The removal of the posterior wall of the pyramid as far as the labyrinth wall follows the radical operation. Starting from the back, the labyrinth is now removed as far as the middle of the horizontal semicircular canal.

One begins by chiseling away the lower vertical semicircular canal, the posterior crus of the upper one and adds to the removal of the posterior half of the horizontal one. The exposure of the posterior part of the vestibule is easy and without danger. Working at the dura of the cerebellum at this great depth is not quite so free from danger on account of the fixation of the dura here to the aqueductus vestibuli. The dura occasionally tears, fluid runs off, and even if the cases mostly take a favorable turn, one cannot be certain of a good result. I have seen such cases end fatally. In order to expose the anterior portion of the vestibule thoroughly, one must remove some bone behind the anterior crus of the horizontal semicircular canal and parallel to it.

The anatomic conditions at the temporal bone are sometimes very complicated, and abnormal position and development of single parts may render the carrying out of this method simply impossible. Such conditions may be seen where the fossa jugularis has developed extremely high up and immediately behind the vertical semicircular canal high inside the pyramid. The disease of the bone in rare cases goes deeper still, so as to compel one to penetrate at the posterior pyramid wall as far as the porus acusticus internus. It seems to me inadmissible to lay down this as a rule, although it has been proposed. We have to remove that which is necessary, not that which is possible. This endocranial method takes place in all complications in the posterior cranial fossa, and in all diseases of the bone which reach as far as the posterior pyramid plane (floor). Here, also, the horizontal semicircular canal, so easily recognizable, serves best as a means of direction and guidance.

It is desirable also expose the anterior portion of the vestibule in such a way that a sufficient view may be obtained from the auditory canal and the treatment thus carried out. The small portions of bone which impede the view from the side and front may be removed without danger to the facialis.

Besides the entire removal of all diseased matter from the portion of the vestibule, this method has the advantage of being absolutely safe for the facialis. If the exposure of the front portion of the vestibule is not carried out exactly from the back as here described, the enlargement of the entrance of the fenestra ovalis downwards and backwards is indicated.

This method is recommended by Neumann as the standard operation in the opening of the labyrinth, and is generally performed. I cannot commend this plan, for the operation is certainly rendered more difficult by the extensive exposure of the cerebellar dura, and involves the danger of perforation of the dura and imminent meningitis. The more rapid healing is of itself no sufficient reason for the regular employment of this method. Healing would take place even more quickly, if the whole of the upper wall of the pyramid were removed.

The depression of the temporal dura at once causes a quite considerable lessening of the cavity of the operation, but this degeneration does not always mean an acceleration of the healing process. It may very frequently have an extremely detrimental effect.

I endeavor to avoid exposure of the dura, if possible, and consider the preservation of the bony edge of the mastoid an advantage well purchased under certain circumstances, even by a somewhat slower healing process. Still I wish by no means to express the opinion that in this way healing is in general delayed.

The semicircular canal method is, in my judgment, very much simpler and easier. On account of pathologic changes, the construction of the temporal bone may be such that this method is by far the easiest.

In a serious destruction of the horizontal semicircular canal, where it is perhaps only necessary to remove 1 or 2 mm. of bony substance for the complete opening of the vestibule, no one will propose the far more dangerous and troublesome, and also technically difficult operation from behind, and if the facialis should have to be entirely destroyed there is certainly no reason for considering this method the standard one.

But there is yet another objection which I should like to raise at this point, and that is as to the name which has lately been given to this method of operation. Ten years ago it was known as my method, later it was called the Neumann-Jansen method, and now is only known in Vienna as Neumann's method.

Although it is a great honor to me that my method should have been adopted by so distinguished a surgeon, and although believing that the opening of the labyrinth is not a difficult feat for another to have done, I consider it my duty to say that I wish to keep the share that I have had in the invention of this method.

OPERATION UPON THE COCHLEA.

Although formerly I proposed the complete removal of the cochlea, and have often performed it, I have restricted more recently myself to the removal of the promontory and the exposure of the lower cochleal whorl for the preservation of the modiolus. I have grown cautious, having seen one case end fatally in which, after complete removal of the cochlea, the dura in the interior auditory canal was perforated by probing, and discharge of the fluid lasted several days.

In order to obviate this with safety, it is necessary to limit oneself to the removal of the promontory, to the exposure of the lower cochlear whorl round about the modiolus, without injuring the modiolus itself. Should one inadvertently or, owing to destruction in the cochlea, arrive at the bottom of the inner auditory meatus, the probing in this dangerous region, and the opening of the dura, must be avoided. Upon the opening of the subarachnoidal space the cerebrospinal fluid appears, mostly to a considerable extent and of several days' duration.

In order to guard against any possibility of lesion to the subarachnoidal space it seems expedient not to interfere with the modiolus at its base, but to leave it here undisturbed. With these propositions of Richards I entirely concur.

When the cochlea in the interior has become transformed into a bed of sequestra, pus and granulations, the removal can be effected without danger by means of the gouge. I generally perform the operation either with a chisel or a burr, the smallest chisel by preference, beginning with the enlargement of the fenestra ovalis towards the front below. If the burr is used, the mode of operation is very simple; it is introduced into the fenestra ovalis, the bone is chipped off towards the front below at the promontory, corresponding to the lower cochlear whorl.

XI.

PATHOLOGY AND BACTERIOLOGY OF SUPPURATIVE DISEASES OF THE TEMPORAL BONE.

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PATHOLOGY.

The subject of this paper is so very large that only a few of the newer phases of the pathology can be mentioned.

In the temporal bone we have to consider the pathology of cavities lined with mucoperiosteum; cavities of varying size and shape with walls of paper thinness to walls of several millimeters thick; mastoid cells filled with air and cells filled with bone marrow. Underlying the mucoperiosteum, we must consider the pathology of the bone of varying character from soft bone to bone of ivory-like hardness. Within the bone we have muscles and nerves; there is the membranous labyrinth; there are blood vessels and the lymphatics—in fact within and bordering the temporal bone (dura and brain) we have to deal with a great number of different tissues. We also have the pathology of wounds to consider. Why one wound heals readily and another refuses to heal. Again, the pathology of the infant varies from that of the adult, as the anatomy varies.

Let us first take up the pathology within the anatomic middle ear and then pass to the pathology of structures without the middle ear, the latter of which are the so-called complications of suppurative diseases of the temporal bone.

I.—PATHOLOGY WITHIN THE MIDDLE EAR.

We are all familiar with the pathologic changes so well described by Körner, Grunert and Bezold in their text books on acute and chronic suppurative diseases of the middle ear, so we will pass on to mastoiditis. P. T. Hald (*Zeit. f. Ohrenh.*, liii Bd., S. 281) has taken the specific gravity of pus in acute middle ear suppurations. He says if the specific gravity is over 1045 one should think of an empyema of the mastoid. From 1046 to 1047 and over the diagnosis is assured.

The Pathology of the Mastoid Cell.—The mastoid cell may be pneumatic or diploetic, containing air or bone marrow, so we may have disease of the mucous membrane extending to the bone, causing an osteitis in the one case or a disease of the bone marrow or osteomyelitis in the other case. Pneumatic cells may be divided by mucous septa, as seen in Fig. 1. In these air cells there are usually several processes going on singly or all at the same time: First, there may be a lack of air; a partial or complete vacuum with its resulting passive hyperemia and transudation. Second, there may be bacterial invasion of the mucoperiosteum with active hyperemia and round cell infiltration (Fig. 2); in infants there may be swelling of myxomatous tissue, so well illustrated by Preysing in "Otitis Media der Säugling." By this process the air space may be nearly occluded. The pavement epithelium is loosened from the submucosa and the exudate may be so great that all of the air space is obliterated, and then, third, pressure begins its destructive work. Pressure shuts off the blood supply and kills the mucoperiosteum and secondarily the bone. The pressure is the greater since it is confined in a cavity nearly surrounded by bone. Besides the pressure we have the toxic effects of the bacteria, which depend on the virulence of the bacteria.

Fig. 3 shows a mastoid cell in an acute suppurative disease of the mastoid, operated on by Dr. E. A. Crockett, of Boston. The mucoperichondrium is strongly infiltrated on the inferior side with necrosis of the mucoperichondrium and hemorrhage into the pneumatic space. Preysing shows in his book some mastoid cells with the vessels in the submucosa filled with thrombi.

Figs. 4 and 5 show pus in cells of a rachitic child who, according to clinical diagnosis, died of diphtheria. The mastoid cells are filled with pus. The mucoperiosteum has been destroyed. Fig. 6, a portion of Fig. 5, shows the destruction of the bone by osteoclasts. The milder types of suppuration with formation of granulations are shown by Preysing. Granulomata may be protective or obstructive in their action, depending upon their position.

Dr. Coutade writes of three cases of *serous mastoiditis* where 15 to 20 cc. of a yellow lemon-colored serum containing some mucus was found at operation. These patients had meningeal symptoms.

Tubercular mastoditis in children. Henrici says less than one-sixth of all mastoiditis in infants are tubercular. There may be a hematogenous primary osseous infection with normal state of the tympanic cavity and antrum, with formation of sclerotic bone and granulations in mastoid cells the size of small peas, and fistulae. The swelling of lymphatic glands over the mastoid is not characteristic of tuberculosis.

Isemer (*Archiv. f. Ohrenh.*, lxvii Bd., S. 97, 1906) reported in detail forty cases, and stated that tubercular mastoiditis occurs in the first half of the years of childhood more frequently than usually supposed; about thirteen per cent of all cases of mastoiditis were tubercular, agreeing with Henrici.

About half of these tubercular mastoiditis cases are primary, that is, originate by the way of the blood channels; the other half are secondary, mostly progressing from tubercular diseased lymphatic tissue in the nasopharynx. The surest proof for a tubercular disease of the mastoid is obtained from microscopic investigations of the diseased parts; less certain are animal experiments. Haike described three cases of primary tuberculosis and two of secondary from the lungs of infants. Paralysis of the facial nerve occurred after a few days with rapid destruction of the temporal bone. In three weeks there were destruction of the ossicles and suppuration of the labyrinth.

In *diabetes* Grossmann says (*Beitr. z. Ohrenh. Festsch. zu Lucae's* 70 Geburtstag, S. 353): "The mastoid is more readily diseased than in those patients without diabetes. In otitis media with diabetes 72.7 per cent had mastoiditis, while of cases without diabetes 56.3 per cent had mastoiditis. The cause is due to the lessened resorptive activity and greater frailty of the mucous membrane; a result of early arteriosclerosis, which occurs in most diabetics together with the altered composition of the body fluids. There is no special form of diabetic mastoiditis."

Wolff (*Zeitschr. f. Ohrenh.*, liv Bd., 1907, S. 322) wrote an interesting paper on *recurrent mastoiditis*. For example, following acute middle ear inflammation, a mastoiditis occurs with formation of abscess; the disease heals after operation completely, but after a longer or shorter intermission in consequence of a new otitis an abscess quickly forms. This process may be repeated several times. The intermission varies from four months to three and one-half years. I have had two cases where there was a recurrence after mastoid operation due to trauma, one playing football and the other struck in playing. Both healed quickly with great retraction of the scar.

Bezold's mastoiditis and *gravitating abscesses* from the mastoid. I have seen a few cases of acute mastoiditis where on operation, the antral cells were macroscopically normal, with the tip cells filled with pus and a perforation extending into the neck muscles. The pus may pass from the tympanic sinus internal to the facial canal to the tip, or the pus may take Voss's route in operative work on the jugular bulb, the infection not spreading by way of the antrum.

Engelhardt (*Zeitschr. f. Ohrenh.*, liv Bd.) in an article on otogenous gravitating abscesses gives eight ways in which retropharyngeal abscesses occur, as follows:

1. Through direct rupture of an osteomyelitic or tubercular focus from the diseased petrous bone to the throat (Muck, Klug and others).
2. Through spreading of inflammation from the middle ear along the tensor tympani or progressing directly in the tissue around the tube (Kien, Haug, Blau, and others).
3. Through the floor of the antrum to the anterior wall of the auditory canal spreading to the retropharyngeal space avoiding or not avoiding the articulation of the jaw (Kien).
4. From the tympanic cavity with resulting perforation into the articulation of the jaw, and from there spreading to the retropharyngeal space (Grüber).
5. From subdural (DeRossi) or extradural (Bramstein) suppuration from the posterior fossa of the skull with resulting perforation.
6. Spreading of sinus thrombosis through the superior and inferior petrosal sinuses to the basillar plexus; secondary suppuration of the same with formation of a retropharyngeal abscess (Schultze).
7. From peribulbar abscesses which spread horizontally under the base of the skull resulting in perforation of the retropharyngeal space (Jansen, Grunert).
8. Through the lymphatics with secondary suppuration of

retropharyngeal glands after acute suppuration of the middle ear (Most).

II.—PATHOLOGY OF STRUCTURES WITHOUT THE MIDDLE EAR.

I can quote only a few facts relating to labyrinthitis. There are many disputed points in this field of otology.

Macroscopic reports of labyrinth suppuration are of little worth; they must be microscopic.

Labyrinthitis in epidemic cerebrospinal meningitis. Göerke (*Verhandl. d. Deutsch. Otol. Gesell.*, xv, S. 227, 1906) found on examining nineteen temporal bones that the internal ear was affected seventeen times; changes were only found in the basal turn of the cochlea. Eleven times infection spread along the acoustic nerve, once along the aquaeductus vestibuli, and three times through the aquaeductus cochleae.

Oscar Wagener (*Archiv. f. Ohren.*, lxviii Bd., S. 272, 1906) considers the aquaeductus vestibuli as a path for infection to pass from the vestibule to the brain. E. P. Frederick in his book, "The Suppuration of the Labyrinth of the Ear," 1905, considers the formation of an empyema of the sac as possible only in extensive suppuration of the labyrinth and only attributes to it the importance of an extradural abscess. Wagener found in the literature twenty-eight cases which had been autopsied; only three had been studied microscopically. In all these cases the labyrinth was found diseased. Politzer reported the only case where there was no doubt. Wagener concludes that the migration of pus from the labyrinth into the sac by way of the aqueduct of the vestibule is excessively rare.

Göerke says that suppuration of the labyrinth extending to the dura can only pass through the nerve canals and the aquaeductus cochleae. The ductus endolymphaticus quickly fills up with newly formed connective tissue. Lange (*Beiträge zur Anat. Physio. Path. u. s. w. des Ohren. der Nase und des Kehlkopf.*, Band 1, H. 1-2, Jan. 20, 1908) found in three cases of severe labyrinth suppuration necrosis of the aquaeductus vestibuli to the aperatura externa, then normal cells and normal saccus endolymphaticus.

Next, we may consider extradural abscess or so-called epidural abscess and subdural abscess or intrameningeal abscess. *Extradural abscess* is a collection of pus between the temporal bone and dura mater. According to Citelli (*Archivio italiano di otologia, etc.*, Bd. xviii, H. 2, S. 151) on otitic extradural abscess he came to the following conclusions:

1. Extradural abscesses are caused as often by acute as by chronic suppurations of the ear.

2. They occur as often in the posterior as in the middle fossa. Grunert says most often in the posterior fossa.

3. True extradural abscess is the rarest of such suppurations.

Subdural Abscess.—A collection of pus between the inner surface of the dura and the arachnoid. Pure subdural suppuration is rare since before pus or pathogenic germs have time to form an inflammatory agglutination on the inner surface of the dura they generally spread over the brain and develop a diffuse suppurative leptomeningitis.

Perisinuous abscess generally forms before sinus thrombosis but may form after sinus thrombosis, causing a renewed temperature and phlebitis.

Kramm (*Zeit. f. Ohrenh.*, Bd. liii, H. 1-2) describes obliteration of the sinus by compression and phlebitis by extradural abscess without an inflammatory thrombosis in children. Leutert does not believe this.

v. Uchermann (*Archiv. f. Ohren.*, Bd. lxxi) on otogenous pyemia and otogenous infectious sinus thrombosis makes the following distinction:

"In otogenous pyemia, so-called, there is no formation of thrombi and fever is caused through absorption of pyogenic bacteria and their toxins.

The metastases are always in the covering of the connective tissue and mucous cavities or in the joint cavities, sometimes in the pleural cavities, not in the lungs. In infectious sinus thrombosis one finds inflammation in the sinus wall with partial (parietal) or complete (obstructing) formation of thrombus. The fever is not necessarily pyemic and becomes so only through suppuration of the thrombus mass and through metastases of the parts to the kidneys, spleen, and to the lungs Otogenous pyemia may start from acute suppurative processes in the tympanic cavity and mastoid cells, sometimes from the labyrinth. Infective sinus thrombosis is mostly from chronic processes. These forms are purely typical. In both forms the picture may change according as the fever has more the character of a poisoning (toxemia) than a pyemia (bacteremia). Such cases are mostly septicemic eventually septicopyemic. In the mixed atypical cases, the sinus thrombosis is parietal. Hessler collected 357 cases of sinus thrombosis, 192 on the right side and 165 on the left. Körner gives as a reason for the greater number on the right side the fact that, as a rule, the right sigmoid fossa is deeper in the bone than the left, and therefore there is a greater surface to be attacked in case of infection. Especially is this the fact when the mastoid is fully developed. Then again Dr. G. D. Cohen-Tervaert (*Archiv. f. Ohren.*, Bd. lxxiv) on sinus thrombosis and jugularis hypoplasia says that Linser found in 1,022 skulls twenty-nine cases of hyploplasia or 3 per cent of all cases. Hypoplasia is five times more frequent on the left than on the right. There may be hypoplasia of the jugular vein and not of the sinus.

Uchermann in Christiana Hospital in thirteen years found thirty cases of otogenous pyemia and infected thrombosis, 9 in children, 21 in adults. The primary disease of the temporal bone was acute in eight cases and chronic in twenty-two cases.

Hessler found in 130 cases, 31 acute and 99 chronic; about the same relation.

In 18 out of the 25 cases reported by Uchermann where sinus phlebitis with formation of thrombus could be proven the suppurative mastoiditis extended to the sinus wall where perisinuous abscess was found in six cases; in seven cases no apparent macroscopic direct communication could be established.

In all five cases of otogenous pyemia without apparent sinus thrombosis, there was no direct union between the suppurative mastoiditis and the respective sinus wall. In one case the pyemia was caused by complicating suppurative labyrinthitis.

In 24 of the 25 cases with thrombus formation the thrombus was situated in the sigmoid sinus, some extending from there posteriorly to the transverse sinus, and some inwardly in the inferior petrosal sinus, and again inferiorly to the bulb of the jugular vein and in the jugular vein itself. In the 25th case, the sinus thrombosis had its seat in the venus plexus around the foramen magnum and further to the occipital sinus, to the torcular, where there was an abscess, and over to the marginal sinus to the jugular bulb. The bulb itself, as also the sigmoid sinus, is free and the point of entrance according to ear diseases (pus in the tip of the mastoid process) is a subperiosteal, suboccipital abscess. In one of the 24 cases the thrombotic formation and softening extended to the torcular herophili to the opposite transverse sinus. The posterior half was filled with pus and the remainder filled with normal blood clot to the right superior bulb of the jugular vein where again there was a pus focus. Such extensions of thrombosis to places where the blood streams are slower or form a rotary motion is also mentioned by Körner. Sinus thrombosis in the bulb of the jugular may be developed by bacteria which pass through the sinus wall higher up and wander into the blood current and not as usual pass in from direct disease of the bone on the floor of the tympanic cavity (Leutert, Körner-Muck)."

"In most cases the cavernous sinus is attacked through extension from the petrosal sinuses or of the venous plexus around the carotid. Through perisinuous abscess and phlebitis of the condyloid emissary veins, abscess formation may occur on one or the other side of the condyloid process around the foramen magnum.

In one instance Jansen (*Archiv. f. Ohren.*, Bd. xxv) reported a case of phlebitis of the mastoid emissary vein extending from a parietal thrombus of the sigmoid sinus.

In the cases where no direct union between the focus in the mastoid process and the diseased sinus wall could be seen, often separated by ivory-like bone, Körner assumes that here also an osteophlebitis is the medium from the small veins of the diploe to the sinus wall.

While the sinus wall in pure otogenous pyemia is normal, thin and of a bluish color, in sinus thrombosis it is more or less thickened, often uneven, greyish or yellowish and of a dirty color. Often one finds it sunken in or compressed by pus and granulations. The thrombus in the beginning is parietal and so impossible to feel. If thrombosis is complete then the sinus feels hard like a cord. The walls may grow together, or the thrombus organize, or the thrombus may rapidly break down and suppurate. The sinus wall may ulcerate in one or many places. If an incomplete thrombus lies under the ulcerated spot, bleeding or aspiration of air may take place. The disintegration of the thrombus does not always take place at the point of infection, but may be further back in the transverse sinus or down in the bulb. Sometimes a fistula forms acting as a drainage tube from the lumen of the sinus.

In six of the twenty-five cases brain abscess accompanied sinus thrombosis."

Postoperative sinus thrombosis is not spoken of in the literature. I have seen one case in which the superior petrosal sinus was wounded resulting in sinus thrombosis and death.

Felix Modowski (*Archiv. f. Ohren.,* Bd. lxxiv, S. 335) writes on the pathology of sinus thrombosis, showing microscopic sections of different parts of a thrombus. The thrombus was organized completely in the left transverse sinus filling it with connective tissue. In the longitudinal sinus the central portion of an organized part had softened.

In general sepsis from the ear, W. Kümmel speaks of neuritis or perineuritis of the nerve roots passing through the jugular foramen especially the glosso-pharyngeus causing pain on swallowing.

W. Cozzolini regards paralysis of the abducens of otitic origin as more of a toxic origin than a circumscribed leptomeningitic focus. Gradenigo believes the paralysis is caused by circumscribed meningitis at the apex of the petrous bone. Transmission of infection from the middle ear is not through the labyrinth, but perhaps by way of the spongy tissue around the capsule. Geronzi believes it to be a reflex paralysis of the neucleus of the abducens.

Kashi (*Archiv. f. Ohren.,* Bd. lxx, H. 1-2) reports 15 cases in infants of otitic dyspepsia with enlargement of the liver. The spleen was enlarged only three times. Pus gets into the digestive tract from the middle ear through the tube. Hartmann and Preysing have described digestive disturbances in infants, as also has B. Gomperz in his book on "Pathology of Middle Ear Inflammation in Infants," 1906. After operation on mastoiditis complicated with syphilis patients are liable to have otogenous pyemia with no visible involvement of the sinuses.

Brain Abscess.—Okada states that in 10 per cent of cases of cerebellar abscess, death occurred before any symptom of intracranial disease was indicated. In 42 per cent the diagnosis was impossible on account of other intracranial complications.

In most cases of brain abscess the bone disease in the middle ear extends to the dura. In about one-half of the cases the dura appears diseased. The diseased dura is nearly always adherent to the overlying brain surface and between the adherent part and the brain abscess the brain substance is a few millimeters thick. Brain abscesses may be single or multiple. Voss (*Zeit. f. Ohren.*, xli, S. 223) believes that the beginning stage of an otogenous brain abscess is a hemorrhagic encephalitis, which goes on to suppuration.

In a compilation of 645 cases of otogenous brain abscess by Heimann,

456 were in the cerebrum, or 70 per cent.
188 were in the cerebellum, or 30 per cent.
1 was in the pons.

Brain abscess occurs three times as often in men as in women, mostly between the second and third decennium.

457 were caused by chronic middle ear suppuration, equal to 80 per cent.

113 were caused by acute middle ear suppuration, equal to 20 per cent.

Otitic Leptomeningitis.—Leptomeningitis is caused by brain abscess, sinus thrombosis, labyrinthitis, unskilled operations as extracting foreign bodies from the middle ear, curetting protective granulations from the tympanic cavity, etc. Zeroni collected 40 cases of post-operative meningitis from the literature and that is far short of the real number. Acute suppuration is quite frequently complicated with meningitis, as is chronic suppuration. From the labyrinth the pus flows through the internal auditory canal and the aqueductus cochleae. Diffuse meningitis gives a positive result for lumbar puncture, i. e., pus, and pathogenic organisms.

Meningitis Serosa.—The cause is not known whether a collateral edema or a beginning circumscribed suppurative leptomeningitis, or whether it is of toxic nature. It was first described by Quincke.

Zeroni (*Archiv. f. Ohren.*, Bd. lxxiii) speaks of *wounds after mastoid operation* which refuse to granulate but keep up a discharge. Sometimes when the diseased focus is curetted the wound granulates and fills up; again, after curetting, the wound only becomes larger and the epidermis from the edges of the wound grows in, since there is no support by granulation under the edges of the wound.

After operation on the mastoid process, regeneration of the bone almost never occurs. Zeroni has examined histologically two cases in which radical operation had been done, one six weeks and the other a year after operation. In the first case there was no apparent new formation of bone. In the latter case there was a thin layer of new bone found, but no osteoblasts were found, so the process could not be studied. Spongy bone has the least inclination to regenerate. In normal cases the wound cavity fills with granulation tissue quickly and forms a hard, fibrous cushion which takes the place of the bone and acts as a protection to any deeper parts as dura. This granulation mass may grow from the bone and also from the edges of the skin wound. Granulations spring from the endosteum of the Haversian canals.

Zeroni describes two types as follows:

First, those in which the granulations develop in great abundance from the edges of the cut wound and so cover the opening, at the same time the process occurs in subcortical layers. The new-formed tissue lies in the cavity and joins the adjacent part as soon as the tampon is removed, forming a beautiful firm, small scar, solid and resistant throughout.

Secondly, in other cases the granulations seem to grow more from within. The filling of the cavity is concentric. The skin edges remain in the position in which they were left after operation. As soon as the underlying cushion has attained sufficient thickness, the skin takes part in the healing process, sending out a thin layer of epidermis. The result in such cases is nearly always a more or less deep depression over the mastoid.

Since the granulations seldom reach the level of the outer skin and because the subcuticle layers fail as in the first type, the skin over such a depression remains thin, and is at first very sensitive. The epidermis may die and a superficial ulcer may form. Later the supporting tissue becomes firm, and improvement takes place, but in consequence of retraction of the connective tissue, the depression grows deeper than ever.

There are many variations from these two types. Sometimes both types take place in the healing process.

The first type is seen most frequently in small wound cavities, the second in large wound cavities, made in laying open extradural and perisinuous collections of pus when an extensive resection of the cortex is necessary to give free drainage.

The edges of the wound must remain open for a long time. Even these large cavities may gradually fill up and form a small smooth scar. In these cases, after a commencing good proliferation, suddenly there is a cessation of the process of healing and the external wound comes to a standstill, remaining wide open and the epithelium grows in to meet the deep granulations.

In the radical operation we used to pack to keep granulation tissue down and it was always a fight. Now we are told to leave all packing out after the first dressing. Sometimes the young granulations are killed after a simple mastoid operation by a too firm packing. The blood supply is cut off and necrosis may result.

BACTERIOLOGY.

Otitis media was first divided into two groups by Zaufal, the so-called genuine otitis and secondary otitis. The genuine otitis occurring after nasopharyngeal inflammation, or tonsillitis or inflammation of the upper respiratory tract. Secondary otitis media occurs after the acute exanthemata and is generally caused by streptococci, but in a number of cases examined by Kümmel the secretions were sterile. Kümmel calls these sterile toxic inflammations.

Kümmel-Süpfles' (Transactions of the German Otological Society, 1907) results on the bacterial investigation of acute middle ear inflammation in Heidelberg were as follows:

"1. Sixty-six per cent were caused by streptococcus pyogenes, about 17 per cent by the streptococcus lanceolatus, about 11 per cent by streptococcus mucosus, and finally about 6 per cent by the micrococcus pyogenes aureus and albus. These results correspond with Denker's investigations.

2. The reason why the streptococcus lanceolatus in the literature on the subject was greatly increased over the present results was that the streptococcus pyogenes and mucosus were not differentiated by the early investigators.

3. The greater number of cases caused by streptococcus lanceolatus occur in the first ten years of life (76 per cent). This is so to a less extent of streptococcus pyogenes; first ten years 44 per cent, the second ten years 22 per cent.

4. The course of the otitis depends on the nature of the inflammatory excitor, in so far as an operation is never neces-

sary in otitis with micrococcus pyogenes aureus and albus, otitis with streptococcus lanceolatus only rarely and under certain conditions in childhood. In otitis with streptococcus pyogenes, operation was necessary in the proportion of 1-3. Twenty-three cases out of 96, otitis with streptococcus mucosus about 1-1.

5. The staphylococcus otitis inclines, so far as the small material allows conclusions, to run a protracted course, and it is possible that the frequent mixture of staphylococci with other inflammatory excitors may be the cause of their protracted course. It is one cause out of others which tends to chronicity,

6. The chronicity of an acute otitis is not necessarily the result of the presence of staphylococci.

7. That the otitis with streptococcus lanceolatus, described "cyclic course," could only be observed in such cases which showed beside the otitis still additional localizations, probably by the same inflammatory excitor.

8. That streptococcus mucosus appears, as with the experience of others, to have a great capability of destroying bone and often appears late in the disease, sometimes even when the otitis has gotten well. This peculiarity was wrongly stated to be caused by the streptococcus lanceolatus by earlier investigators, because they had no means to differentiate it from the streptococcus mucosus.

9. The course that the disease takes depends not alone on the kind of inflammatory excitor, but also on the peculiarities which lie in the patient himself, of which we only know the smallest part.

10. One of these peculiarities is the individual nature of the union between the epitympanum and mesotympanum. A free union between the two gives a better prospect of the otitis getting well without operative procedure. The difference in the course of the disease between the so-called epitympanal and mesotympanal otitis is brought out most clearly in cases caused by streptococcus pyogenes, in so far that almost all of the cases which come to operation are of the epitympanal type. A sure pathologo-anatomic support for this separation fails as yet.

11. Moreover, it appears as if the way of invasion of the inflammatory excitor according whether it is termed a salpingogenous or hematogenous otitis, influences the course of the disease, in so far as the hematogenous infection presents more

the picture of a severe general disease and therefore the cavities of the mastoid process take on a less intense sympathetic affection. Our knowledge in relation to the way of invasion of the inflammatory excitor is still, however, insufficient.

12. So-called secondary otitis can apparently exist sometimes without participation of microorganisms, apparently through toxic action and the otitis either quickly heals without the entrance of organic inflammatory excitors or subsequently become infected with such and then runs as other cases of otitis."

Denker (*Verhandlung der Deutch. Otol. Gesell.*, 16 Versammelung) on bacterial investigations in acute middle ear suppuration examined 29 cases of acute empyema of the mastoid process. The diplococcus pneumoniae wholly failed throughout the 29 cases, although 9 of the children had pneumonia. Kümmel-Süpfle found pneumococci only in two cases where the pus was taken from the mastoid.

Wittmaack (*Deutch. med. Woch.*, Nr. 31, 1906) found that staphylococci were only found in the secretion when the secretion had continued several days, therefore staphylococci infection is probably secondary.

Chronic otitis is due to those bacteria which cause a protracted course as streptococcus mucosus and in cases of mixed infection.

Proteus vulgaris is rarely mentioned in acute suppuration. Lauffs (*Archiv. f. Ohrenh.*, Bd. lxx, S. 187) found proteous vulgaris often accompanying cholesteatomata. It often destroys the cholesteatomata and then this putrid bacillus has a good chance to infect the cranial cavity. This bacterium generates sulphuretted hydrogen, ammonia, and indol.

Brieger (*Verhandlung der 7 internat. otol. Kongress*, 1904) said that microorganisms within the middle ear space which are saprophytes could develop under favorable circumstances where the air was occluded as in the labyrinth or arachnoid space and become virulent, causing meningitis.

Rist (*Zentralblatt f. Bact.*, Bd. xxx) says as a result of numerous investigations that he has never found a single fetid suppuration without finding anaerobic bacteria. Franz Kobrak (*Archiv. f. Ohrenh.*, Bd. lxxiv) has an interesting paper on the importance of anaerobic bacteria for the existence of sinus thrombosis and reports two cases.

Pseudo-diphtheria bacilli are more frequently found in chronic than in acute inflammation. They can only be differentiated from the true diphtheria bacilli by experiments on animals.

Lermoyez believes that there are osteophile streptococci.

H. I. Wolff (*Zeit. f. Ohrenh.*, Bd. liv) on the cause of recurrent mastoiditis gives three methods by which the mastoid may become infected:

First, the retention of latent germs in the mastoid process.

Second, a new infection of the mastoid process from the tympanic cavity by virulent germs.

Third, a new infection by mild virulent germs which can cause abscess due to the lessened resistance of the tissue after the first disease and operation, if at the same time there exists a bad general condition or a disposition to otitis.

Ernst Leutert (*Archiv. f. Ohrenh.*, Bd. xiv) in a paper on "The Diagnosis of Otitic Sinus Thrombosis by Means of Bacteriologic Investigation of the Blood," comes to the following conclusions:

"1. The presence of streptococci or other microorganisms occurring in the blood of the sinus in considerable number and at the same time sterile blood in the arm indicates a diagnosis of sinus thrombosis.

2. The diagnosis is sure if the blood in the arm gives a positive result. In cases of positive results in the blood of the arm the prognosis is unfavorable since it must indicate a lessening of the bactericidal strength of the blood.

3. If the sinus blood is investigated alone and a moderate number of pyogenic microorganisms appear, then there is thrombosis unless taken shortly before the patient's death, in which case other septic disease may flood the whole blood system with microorganisms.

4. The negative result of the bacteriologic investigation of the blood in the vein of the arm proves nothing against the presence of a septic thrombosis, for in recent cases the microorganisms are killed by the bactericidal action of the blood.

5. The condition of microorganisms getting into the blood through permeability of the sinus wall. If sinus phlebitis is the first stage of thrombosis these cases must be studied further, since in such early cases the unweakened bactericidal action of the sinus blood has given negative results or only rarely colonies. The number of cases is too small to draw conclusions."

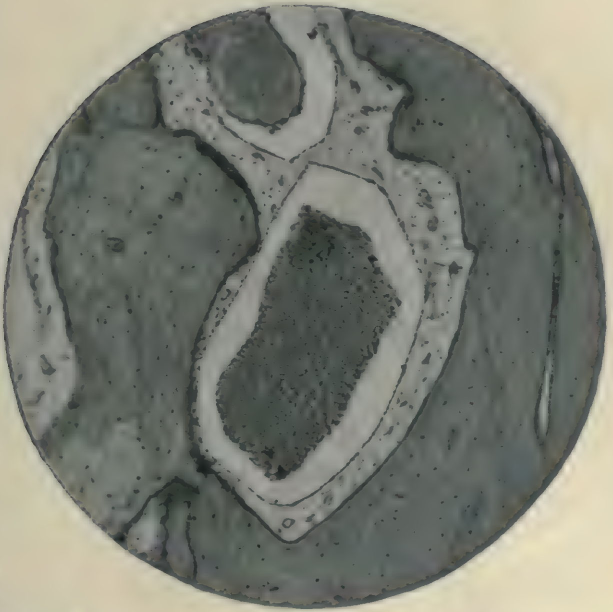

FIG. 1.—Pneumatic cell of mastoid divided by mucous septum.

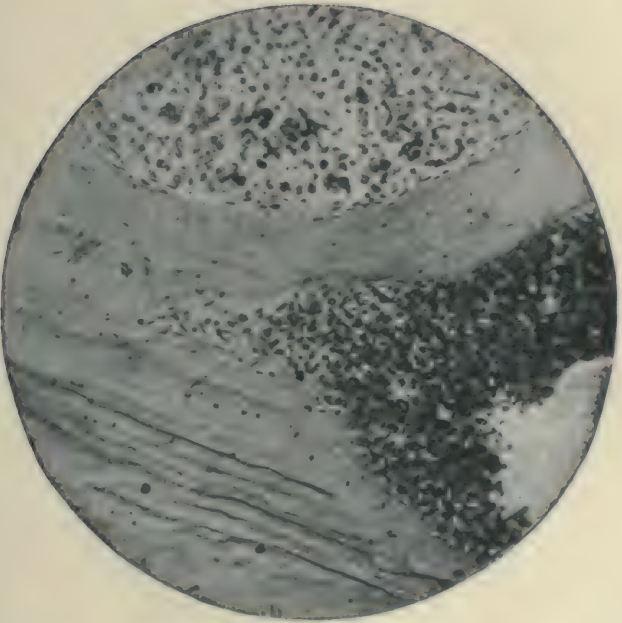

FIG. 1A.

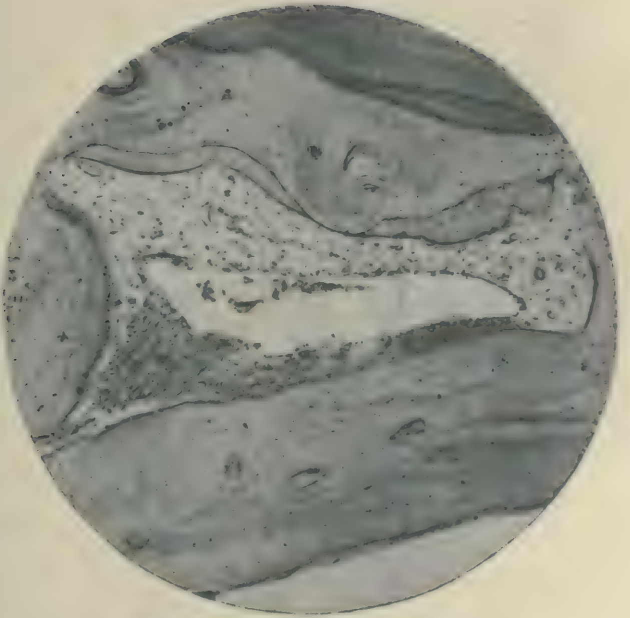

FIG. 2.—Round cell infiltration.

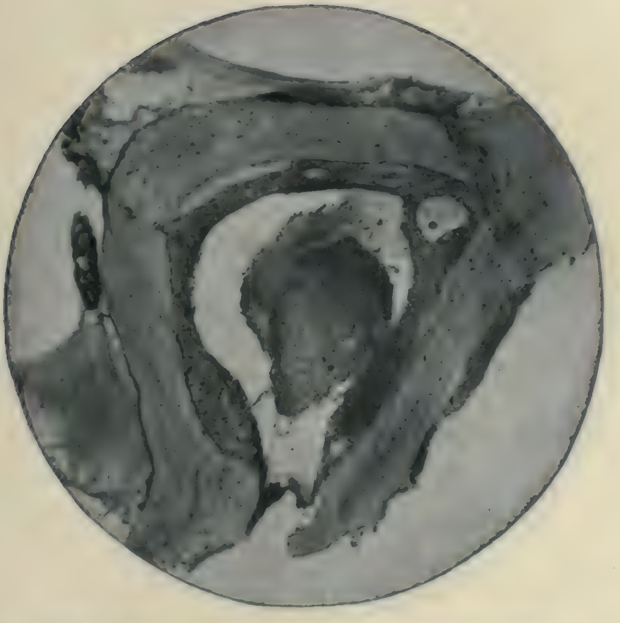

FIG. 3.—Infiltration; hemorrhage; necrosis.

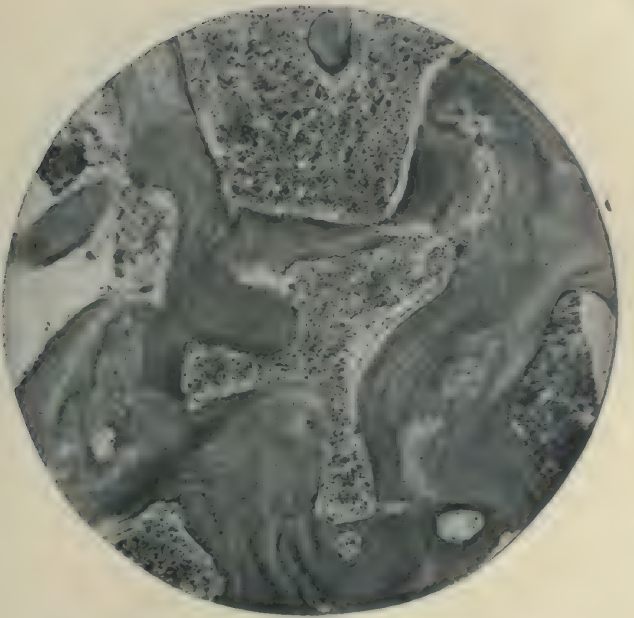

FIG. 4.—Pus; death of mucoperiosteum; caries.

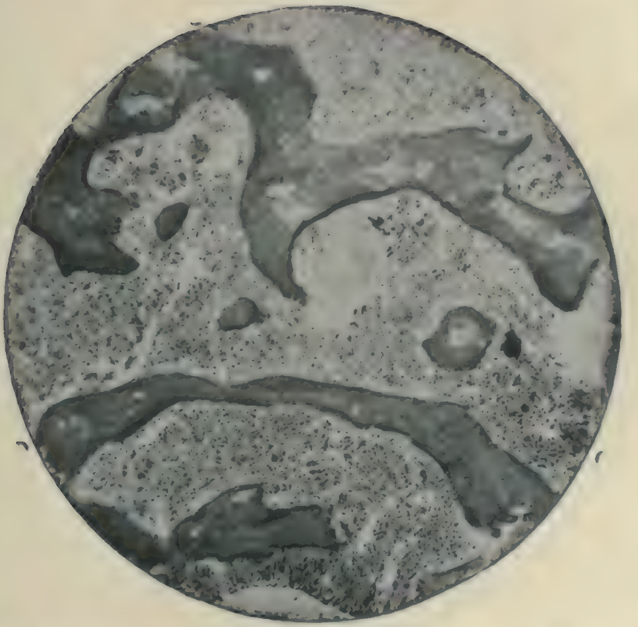

FIG. 5.—Mucoperiosteum destroyed, showing pus in mastoid cells and necrosis of bone.

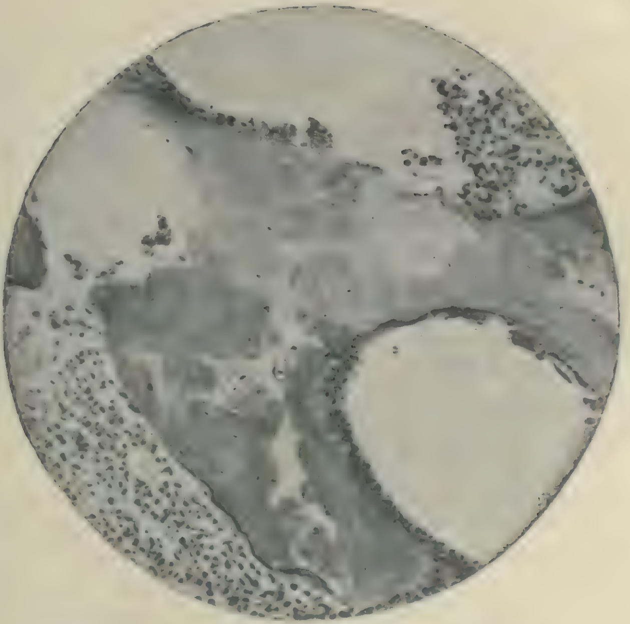

FIG. 6.—Destruction of bone by osteoclasts.

XIII.

OPERATION FOR BRAIN ABSCESS OF OTITIC ORIGIN.

BY J. A. STUCKY, M. D.,*

LEXINGTON, KY.

I wish to apologize to the section for not having prepared a formal paper with some care, but really there is a good excuse back of it. Those of you who have seen the last issue of *The Laryngoscope* will find in that journal, if you care to read it, some of my ideas on this subject. There are some points I want to go into with a little more detail. My topic is "The Operation for Brain Abscess of Otitic Origin."

In submitting a paper on this subject, the supposition is that the diagnosis has been made and that a brain abscess is present; and yet the diagnosis is so often uncertain, on account of the absence of focalizing symptoms and pathognomonic indications that it is well to pause here and consider them carefully, I very much doubt whether in many cases we have any absolute indications of brain abscess. In every case of chronic suppuration of long standing, in which there is marked evidence of septic poisoning, we should be prepared in doing a radical mastoid operation to deal with an extra- or subdural abscess. This point I want to emphasize. In chronic abscess sometimes the abscess is encysted and few symptoms exist. The most prominent of those of an abscess of the brain may be pain in the head, vomiting, with little or no nausea.

I have had only twelve or thirteen cases of brain abscess and I wish to refer to the two most frequent symptoms, and mention the treatment I have followed in these cases. There was a slow pulse and a normal or subnormal temperature. One case upon which I operated on three days ago had a temperature of 96°, and a pulse of 63. The child was twelve years of age. She had an occasional rigor, with some intraocular changes, choked disc, but these symptoms are not reliable. The discharge from the ear had stopped suddenly two weeks ago and the child began to decline. Cerebration became slow, and she had some of the symptoms that were described by Dr. Layman this afternoon in connection with his case. Mental apathy was marked. I found occasional paralysis of certain groups of muscles. Fortunately, we have in lumbar puncture in these cases a valuable aid in diagnosis. If the fluid withdrawn shows marked infection, we are justified in supposing that we have a brain abscess, if in addition there are any marked meningeal symptoms.

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*Stenographic report of paper.

One peculiar feature about the cases I have had is the description of the pain given by the patient. Several have said it is so deep that they cannot find it. There is no pain apparently over the mastoid, but they describe it as being "away down in the ear, so that you cannot touch it; it must be an inch or two in there," and it may be an occipital or temporal pain. I have found in these cases, notwithstanding the experience of Dr. Day, that the differential blood count was most valuable, and I have had examinations made frequently, every eight or ten hours, especially in the fulminating cases.

The prognosis, of course, is to be guarded. Usually it is unfavorable.

Of all cases we have to deal with, those in which we suspect brain abscess are the ones in which we ought to "carefully box the compass," so to speak, and take our bearings. We should consider most carefully the excretory and secretory condition of the patient and the powers of resistance—a point which I emphasized in my address at Louisville. Indican in the urine should be the cause for more anxiety than the presence of either albumin, sugar or casts, and in all these chronic suppurative conditions of the ear, I know of none in which intestinal metabolism is more interfered with or where the vicious circle gives us more trouble. I know of nothing where peristalsis is so completely paralyzed as it is in these cases. You may treat it with purgatives, but what will purge a patient ordinarily will not purge a patient with meningeal irritation or brain abscess; you can give them ten times as large a dose. So I am very careful to make a special effort to get elimination and secretion started before beginning any operation where I expect to go into the brain.

The method of operating is a little different from that usually described. First, I do as thorough a radical tympanomastoid exenteration as is possible for me to do; then I remove all the tegmen antri and tegmen tympani until I get the floor of the middle fossa well exposed; as complete an exposure as desired may be secured without making a counter opening with the trephine in the squamous portion by widening it until it is sufficiently enlarged. If the dura is discolored and bulging, I open with Bier's knife at the point where there is the most distension, and then, instead of searching with a knife, I invariably use Jackson's searcher. I believe this is safer than using the knife. The different parts of the brain may be penetrated to any depth and opened, and if there is any pus there, it will escape. The incision may be enlarged, if necessary.

In this connection I wish to speak of a very important point which Sir Victor Horsley presented to the British Medical Association a year ago, about which I have grown enthusiastic. His method, which I use in all radical operations in which I expose the sinus or dura, is the almost constant use of hot normal saline solution, at a temperature of from 115° to 120°, never lower than 115°, never higher than 120°. Here is where operative technic counts for more than in any other operation in surgery. Every man and woman connected with it has something to do, and should know how to do it, and when to do it. One nurse takes care of the normal saline solution and must have nothing else to do. The thermometer is always in the douche bag. If the water is below 115°, it does not control hemorrhage; if above 120°, as Sir Victor Horsley has demonstrated, there will be some little reaction, but between 115° and 120° all oozing can be effectually controlled. In addition to this, all soft parts are kept moist and clean. All bone dust and all exudative material are washed out.

After making an incision and beginning exploration, I practically operate under a small stream of water. The hot saline solution is allowed to run from a medicine dropper, attached to the douche bag, over the wound, and the parts are always warm, moist, and clean. If an abscess is found and the incision is enlarged, I do not believe in making any exploration with the finger; if any cheesy particles of brain matter are found they are to be clipped off carefully or sponged away. The sponges should always be wet with the normal saline solution.

After finding the abscess and evacuating it, we should be extremely careful in washing it out. The force of the normal saline solution should not be any more than enough to throw a stream six or eight inches. I do not hesitate to put a pipette or medicine dropper into the opening and irrigate it until it becomes clean.

I think the most important point to be observed after the operation is the after-dressing, which should maintain constant and thorough drainage. I have ceased to use the cigarette drain or any gauze. I always have on the table three sizes of tubes of flexible rubber, perforated size (see figure). These I cut to the required length. If it is a sphenotemporal abscess, the tube should be placed in the opening through the dura, with simply a little drain of narrow wick of gauze in them, but not enough to plug it up. If in the posterior fossa it should be placed in the proper position, and let it come out back of the ear. The other tube goes up through the auditory canal just as I drain in the radical mastoid operation. A dressing of bichloride gauze is then applied and moistened every four hours with the 1 to 3000 or 5000 solution of mercury bichlorid. The dressing is changed every twenty-four hours under the same aseptic precautions as those under which the operation is performed, because the moment the gauze in the tube becomes saturated or a little clot of blood forms in it, it ceases to act as a drain and becomes a cork. The gauze is put inside the rubber tube and allowed to project, on the order of a siphon. One of the chief things is the thorough drainage we get, and this is maintained by daily dressing the wound. In my first few cases I left the dressing on for two or three or five days, and I believe some of the bad results I had were due to that. I treat my radical operation cases on the same principle. Next to the sponging and drainage in the operation I believe that the quantity of the anesthetic cuts a great figure. It is astonishing how little of the anesthetic we can get along with in these brain cases and in radical mastoid operations. I have had patients held for an hour and a half to two hours on surprisingly small amounts of ether, but not without the patients squirming a little. I think some of our bad results are due to the fact that we have given the patients too much of the anesthetic. After the operation the question of nourishment is no more important than that of elimination for the first week or ten days.

XIV.

CAUSATION OF THE SUPPURATION OF THE TEMPORAL BONE.

By William E. Sauer, M. D.,

St. Louis.

The vast majority of suppurations of the temporal bone are secondary to an inflammatory process in some part of the auditory apparatus. In discussing the causation of these suppurations, I will take up the etiology of acute middle ear suppurations, chronic middle ear suppurations, mastoiditis, and the suppurations of the labyrinth.

While it is admitted by some that a purulent exudative process may exist without the presence of pyogenic bacteria or at least without its being possible to demonstrate their presence, it may be safely stated, that practically all suppurative conditions of the temporal bone are due to the invasion and action of pathogenic bacteria. The bacteriology of the subject is to be taken up in the paper that is to follow. Therefore, I will deal only with the mode of invasion and the predisposing causes.

The invasion of the middle ear may take place through the Eustachian tube, through the external auditory canal, by way of the blood or lymph channels, by way of the labyrinth and along the sheaths of the auditory and facial nerves, and from traumatism.

Invasion through the Eustachian tube is the most frequent. Infectious material may be carried by direct extension from continuity of tissue, or may be forced into the tympanum by air or fluids. Inflammatory conditions of the nose and nasopharynx frequently extend through the Eustachian tube into the middle ear by continuity of tissue. This extension of an inflammation or the invasion of the middle ear, is favored by the presence of nasal and nasopharyngeal obstructions with their attendant condition of persistent congestion of the mucous lining of the tube and typanum. These obstructions may not only interfere with the normal ventilation of the tympanic cavity, thus retarding or obstructing the venous circulation, with resultant exudation into the middle ear cavity. This exudation may become infected, as under these circumstances it offers a favorable medium for the development of pathogenic microorganisms, which, according to Hasslauer, may be present even in the normal middle ear, or which may have gained entrance from the pharynx.

The infection of the middle ear by the air, may take place when attempts are made to clear the nasal passages by forcibly "blowing the nose" when the nose or nasopharynx contains infectious material. This may also occur through the use of the Politzer method of inflation, or when the catheter is employed. Infection through the medium of fluids entering the middle ear through the Eustachian tube, occurs most often from the faulty methods in using the nasal and postnasal douche; through the practice of snuffing up fluids into the nose, or while bathing some of the fluids may accidentally enter the nose and mouth, and find its way into the Eustachian tube. This, I have found to be a rather frequent cause of suppurative otitis media; and in most cases it was determined that the patient had visited one of the indoor swimming pools.

There are a number of acute suppurations of the middle ear which have their origin in the nasal cavities, and which are the result of operative interference. During the period when the electrocautery was so indiscriminately employed in the nose and pharynx, acute middle ear suppurations were frequent sequels; and even to-day this may occur in spite of all precautions. Packing the nasal passages after operative procedures may also be the cause of an acute otitis media; especially if the tampon is allowed to remain in position for too long a time. I have known of a number of instances where an otitis media followed the removal of adenoids and tonsils.

Infection of the middle ear through the external auditory canal, may occur as a result of solution of continuity of the drum membrane, either by traumatism or by the action of an inflammatory process, or through the Rivinian segment. Traumatic perforations, the result of direct or indirect violence, with primary or secondary infection, are not infrequent sources of middle ear suppurations; otitis externa with its extension, according to Dench, through the Rivinian segment, is also a cause of otitis media. More rarely, the development of a vegetable fungus in the auditory canal may lead to a destruction of the drum membrane, with secondary involvement of the middle ear. The contact of cold water or cold air if continued for a sufficient length of time, may cause an inflammation of the drum membrane with secondary middle ear involvement. Hot water or other irritating fluids poured into the external auditory canal, may produce the same result. Unskilled efforts at removing foreign bodies from the external auditory canal, have resulted in injury to the drum membrane with infection of the middle ear.

Infection of the middle ear by way of the blood and lymph channels, may take place in diphtheria, measles, scarlet fever, tuberculosis, influenza and endocarditis. The infection of the middle ear through the labyrinth or by way of the facial and auditory nerves occurs very rarely, but it may occur in cerebrospinal meningitis or from a meningitis from other causes.

Acute suppurations of the middle ear occur most often in children. The underlying factors accounting for the prevalence of the acute middle ear suppurations in children are, according to Kopetsky:

1. Constitutional diseases, and the hereditary influence of tuberculosis and syphilis.
2. The prevalence of the exanthemata during childhood.
3. Anatomic structures peculiar to childhood in the ear and its adnexa.

Among the general diseases given as predisposing causes are, rachitis, bronchopneumonia, the gastrointestinal derangements, or any disease which may weaken the body resistance. Kopetsky with Koerner holds that predisposing factors are tuberculosis or syphilis in the parent; the children themselves not necessarily being either tuberculous or syphilitic.

Primary tuberculosis of the middle ear is a comparatively rare affection. It is most often encountered as a primary disease in the mastoid. A secondary invasion of the middle ear through the Eustachian tube is not infrequent in older patients, and is seen most frequently in patients with pulmonary tuberculosis; especially during the last stage.

Syphilis, according to Politzer, may also be the cause of a middle ear suppuration; the specific virus entering the middle ear through the Eustachian tube. It occurs most often as a consequence of a syphilitic nasopharyngeal affection which directly involves the tube.

The exanthemata are among the most frequent causes of acute middle ear suppurations, and of these, scarlet fever is the most frequent and destructive in its effect. According to the views of the majority of investigators, the otitis is most often of a secondary nature, and the infection enters through the Eustachian tube, though it is not impossible that the specific inflammation may involve the pharynx, tube, and tympanum synchronously. In some cases the pharynx is not involved, showing that the infection enters by way of the blood or lymph channels. Levy describes a case of scarlatinal otitis which appeared seven days prior to the eruption. In spite of an early paracentesis the mastoid became involved and operative interference was necessary. The otitis media that occurs in the later stages of scarlet fever runs a much milder course. According to Bezold, one-half per cent of all ear diseases are due to scarlet fever; and in from 20 to 30 per cent of all cases of scarlet fever there is an involvement of the middle ear.

Measles is considered as the next most frequent cause. The characteristic inflammatory macules (in the early stages of the disease) have been found on the tympanic mucous membrane; coincidently with their appearance in the mouth and nasopharynx, and so are not secondary to an inflammatory condition of the nasopharynx. Later in the disease, an infection may take place as the result of an inflammatory process in the nasopharynx.

Diphtheria is also a frequent cause of middle ear suppurations. It rarely occurs as a primary affection of the middle ear. According to Hasslauer, there have been only three bona fide cases reported; namely, those by Krepuskas, Lewin, Gruenert and Schilling. The other cases occur as a secondary involvement through the Eustachian tube.

Influenza is also a frequent cause of acute suppurative otitis media. The infection may enter by way of the blood or lymph channels, and the otitis occur during the first or second day of the disease; or it may occur later, as the result of an infection entering through the Eustachian tube after the disease has practically run its course.

An acute suppurative otitis may also be the result of the direct extension of an inflamed parotid gland, as it occurs in mumps. This extension occurs through the glaserian fissure; or it may occur secondarily from the inflammatory affection of the pharynx and nasopharynx, which sometimes occurs in this disease.

According to Bezold, acute otitis media occurs in from 2 to 4 per cent. of all cases of typhoid fever. The ear involvement occurs in the fourth or fifth week, and may be the result of a pharyngitis or of septic material being blown into the ear in an effort at clearing the nasal passages; or the infection may enter by way of the blood current from an endocarditis or from collections of pus in some other part of the body.

The anatomic structures peculiar to children, which, according to Kopetsky, are factors in tending to cause the prevalence of middle ear suppurations are: the adenoid tissue, the short and wide lumen of the Eustachian tube, and the embryonal character of the contents of the middle ear. He considers that the normal adenoid acts as a barrier to the entrance of particles of food into the Eustachian tube in infants. On the other hand, the effect of the diseased or hypertrophied pharyngeal tonsil with its secretion of ropy mucus and resulting congestion of the pharynx, offers an attractive place for the development of pathogenic microorganisms. Aside from the local effect of obstruction, their influence on the general economy by reducing its resistance, makes the presence of adenoids a most potent factor in the causation of middle ear suppurations.

All writers agree that the widely open lumen of the Eustachian tube, as well as its shortness and horizontal direction as present in children, favors the invasion of infectious material into the middle ear. The retrogressive changes that take place in the embryonal tissue in the middle ear of infants, also furnish, according to Weiss, Preysing, Kopetsky and others, soil for the development of pathogenic bacteria.

Chronic suppurative otitis media is usually regarded as a sequel to acute suppurative otitis media. An exception occurs in tuberculosis; as in this affection the process may appear with a chronic character from the beginning. Just when an acute case passes into a chronic one, is not easy to determine; but after a suppuration has continued for two to three months, it is usually regarded as chronic. The general conditions that influence or cause the transition of an acute into a chronic case are, constitutional diseases which tend to lower the resistance of the patient, such as tuberculosis, syphilis, anemia and marasmus. The conditions which are important local causes of middle ear suppuration are, according to Politzer, 1. The granulations and polypoid growths of the mucous membrane of the tympanic cavity and on the membrana tympani, which develop in the acute stage. 2. The retention and caseation of the purulent secretion in the lateral concavities of the tympanum, in the depression of the petrous bone, and in the cells of the mastoid process. 3. Caries which is developed in the mastoid or in another portion of the temporal bone during the acute course of the disease. 4. Chronic inflammations of the external ear, as is sometimes the case in erysipelas and chronic eczema. 5. Chronic blennorrhea of the mucous membrane of the nasopharynx and ozena.

Just what part the microorganisms play in the development of a chronic suppurative otitis is not definitely known, though an otitis occurring during the course of scarlet fever and diphtheria is more apt to be chronic. There can be no doubt that a number of acute cases become chronic as a result of improper treatment and unhygienic surroundings.

Inflammations of the mastoid process may occur during the course of an acute or chronic middle ear suppuration; or they may be primary and the result of traumatisms. The inflammation of the mastoid occurring during the course of acute chronic middle ear suppurations is due to insufficient drainage, either because of the opening of the drum membrane being inadequate for the accumulated discharge, or from a blocking off of the communication between the mastoid cells and the middle ear. If the views of Politzer are accepted, the mastoid process contains pus in every case of acute suppurative otitis media, and so long as the communication between the antrum and the mastoid cells is free, stagnation of the secretions in the mastoid does not take place, and no reactive symptoms are produced; however, it can be easily understood that if the opening is small, a swelling of the mucous lining of the cells alone may produce an occlusion with stagnation, and as a result, the osseous tissues may become involved. Other causes which tend to mastoid involvement during middle ear suppurations are; exposures to wet and cold, the forcible injections of fluid into the ear, or the employment of the air douche. The experiments of Schutz on the cadaver, have proven that the employment of the air douche in an acute otitis media when the perforation in the drum membrane is small, is a dangerous procedure, as some of the pus may be forced into the mastoid cells. In seventeen of the eighteen cases on which experiments were made, when the perforation in the drum membrane was small, the stained pus placed in the middle ear through this small opening, was found in the remotest cells of the mastoid after catheter inflation, but he found the pus in only one of sixteen in which the same experiment was made in the presence of a large opening in the drum membrane. The occurrence of mastoid involvement is frequently seen in certain infectious diseases, as in scarlet fever, measles, diphtheria and influenza. The conditions which favor the extension of a chronic middle ear suppuration into the mastoid cells are; narrowing of the external canal, the presence of polypi and granulations and the formation of cholesteatomatous masses, as these interfere with drainage and lead to caries and necrosis of the bone.

Primary inflammations of the mastoid are mostly the result of traumatisms. Tuberculosis is, according to Henrici a comparatively frequent cause in children. He found that one case out of every five mastoid cases operated upon by him, were tubercular; and he believes that the majority were primary. Isemer believes that 13 per cent. of all mastoid cases in children are tubercular, and half of those are primary. Haike demonstrated that out of five cases of tuberculosis of the mastoid examined by him, three were primary.

It is possible that a number of so-called cases of primary mastoiditis are really secondary to a middle ear involvement, so slight as to be overlooked.

The invasion of the labyrinth may take place through the middle ear, by way of the blood and lymph channels, from the cranial cavity by way of the aqueducts, the facial and auditory nerves, and through trauma. The great majority of labyrinth suppurations result from the extension of middle ear suppurations. In the acute otitis media due to scarlet fever, measles, diphtheria and influenza, such extension is more apt to occur. In the chronic cases, tuberculosis and cholesteatoma play an active part. This extension may take place through the anastomosing vessels of the middle ear and labyrinth, or through a destruction of the bone from inflammatory processes. The labyrinth capsule may also be opened during operation on the middle ear, or penetrated by foreign bodies entering through the middle ear. According to Hinsberg, the most frequent paths of entrance for this infection, are the round and oval windows, the promontory and the horizontal semicircular canal.

The invasion of the labyrinth by way of the blood and lymph channels, as it occurs in mumps, and the invasion from the cranial cavity by way of the aqueducts and nerves, as well as in fractures of the temporal bone, are less important from a surgical point of view.

Regarding the frequency of labyrinth suppurations, Friedrich states that the labyrinth is involved in about 1 per cent of all the cases of middle ear suppurations; but as our knowledge of this subject increases, it will be found that the percentage of labyrinth suppurations will possibly be increased.

Humboldt Building.

XV.

A REPORT OF ATYPICAL AND TYPICAL CASES OF SINUS THROMBOSIS COMPLICATING MASTOIDITIS.

By W. R. Dabney, M. D.,

Marietta, Ohio.

The more cases of sinus thrombosis that I see as a complicating factor in mastoiditis, the more I become convinced that the symptom complex, as laid down in the books,—such as chills, hyperpyrexia, sweating, and a rapid easily compressed pulse,—is not always diagnostic of a septic thrombophlebitis.

There are a number of acute infective conditions that are quite frequently manifested after mastoid operations which so closely resemble sinus thrombosis in their early symptoms, that the diagnosis should be made by exclusion, rather than by the symptoms of sinus infection that may be presented.

Pneumonia is not infrequently seen as a complication after operations upon the temporal bone; that this disease can very closely resemble a sinus infection—in its symptomatology—is well illustrated in the following case:

Case 1.—Child of 12 years. An old chronic otitis media exacerbated by an attack of tonsillitis; operation on tenth day after mastoiditis began. At time of operation the temperature was 99° F. and pulse 100.

Operation.—Subperiosteal abscess over mastoid and temporal fossa; mastoid empyema extending into the superior meatal cells and cells of the anterior wall of the bony canal; perisinuous extradural abscess over the arch joining the horizontal and vertical portions of the lateral sinus.

The sinus was not explored as the history of the case was not very clear, and the patient was not taking the anesthetic kindly.

The posterior bony canal wall was taken down and Panse flaps stitched into the mastoid cavity. The vertical incision over the mastoid was left open.

Six hours after the operation, patient had a chill which was followed by a rise of temperature to 105° F. and pulse 150. There was frequently repeated slight chills followed by high temperatures and rapid pulse for two days when the characteristic sputa of pneumonia appeared and rendered the diagnosis clear. Auscultation and percussion were negative during this time, as is often found in the early stage of a deep seated central pneumonia.

The pneumonia resolved by lysis, in this case, and the patient was discharged at the end of the sixth week after the mostoid operation.

Acute septic nephritis is another complication that should be considered before it is determined that an infected clot is present in one or more of the venous sinuses of the dura mater.

Case 2.—Purulent mastoiditis in a young man complicated by extradural abscess above the roof of the antrum and tympanum.

Three days after operation, there was a chill, rapid rise of temperature from 99° F. to 104° F., and exhaustive perspiration. The pulse increase was out of ratio to the temperature as it was never above 85 per minute; there was partial suppression of urine and the urinary analysis showed albumin, pus, and cells from the pelvis of the kidney.

The kidney complication cleared up under appropriate treatment.

Case 3.—Child 14 years of age. Acute mastoiditis; ear had been discharging for six weeks.

At the operation, we found only a small quantity of pus and thick bloody serum, in abundance, in all of the cells of the mastoid.

On the sixth day following the operation the patient had a chill which was followed by a rise in temperature and pulse, and there was free perspiration. The next day there was seen a slight croupous exudate on the same side of the throat as the operation. There was tenderness along the upper third of the internal jugular vein, which was no doubt due to the involvement of the anterior cervical lymph nodes that overlie the internal jugular, although there may have been a phlebitis of the jugular by contiguity of structure.

A culture from the throat showed the influenza bacillus.

Under aconite, quinin, camphor and aspirin, the symptoms of sinus thrombophlebitis disappeared.

We have seen a number of cases of gastrointestinal disturbances following mastoid operations, which upon a superficial observation could easily be mistaken for sinus infection, but we have only to make the test for indican to clear up the diagnosis; when it is found, the case is to be treated accordingly and the symptoms of sinus involvement will disappear with surprising rapidity.

Pulmonary, kidney and liver complications as concomitants of an acute osteomyelitis of the temporal bone, are easily accounted for, when we consider the fact there is, in this condition, a thrombosis of the arteries as well as of the veins, and when small particles of these thrombi become detached they are deposited as infected emboli in the above mentioned organs, and thus form a nidus of infection that gives rise to the symptoms which indicate that these organs are involved in a septic process.

Case 4.—White female, age 14 years. I was called to see this patient on account of an acute exacerbation of a chronic purulent otitis media.

History.—Both ears had been discharging at intervals for ten years. During the apparent quiescent periods, the patient would remove scales of inspissated pus from her ears; has had most of the diseases incident to childhood, and her ear trouble had its inception in an attack of scarlet fever.

Present illness began a week ago during an attack of pharyngitis; ears began to discharge on the second day; the flow from the left ear was very profuse while that from the right ear was scant and foul smelling. During the evening previous to my first visit she had chilling of the lower extremities that was followed by gentle perspiration.

Present Condition.—Temperature, 100.6° F.; pulse, 120. Complains of intense paroxysmal pain in the right ear, the pain being somewhat relieved after there is a free gush of pus; tenderness to pressure applied over the antrum, tip, posterior meatal cells, and mastoid emissary vein; skin over mastoid is glazed and reddened; postauricular groove is obliterated; large perforation in the lower posterior part of drum; decided bulging of inner end of posterior-superior membranous canal.

The streptococcus was the predominating organism in a smear that was taken from the pus as it came through the perforation in the drum.

Operation advised and declined as the father of the child was out of the city and the consultant did not agree with me as to the advisability of an early operation.

December 14th.—Saw patient at 1 and 9 p. m., and the objective symptoms were more pronounced although she said that she felt better. When operation is proposed it usually has this effect.

December 15th.—Word was received from the father that we operate at once if it was deemed advisable. As the child had had several slight chills during the night and was prevented from sleeping by severe pain in the ear and mastoid, it was thought best that we operate as soon as she could be prepared.

Six p. m. operation; chloroform; the mastoid was densely sclerosed and not a single cell was encountered until the antrum was reached when pus and granulations as well as cholestoatoma were encountered; antrum was larger than usual and the walls were undergoing ulcerative degeneration; the posterior canal wall was chiseled away and the malleus and incus, which to the unaided eye appeared to be normal, were found imbedded in a mass of fibrous connective tissues that had formed between the drum and the inner tympanic wall; this condition in connection with the granulation formation in the antrotympanic duct prevented free drainage from the antrum into the middle ear and accounted for the paroxysmal pain as the antrum was not emptied until the pressure became great enough to force the pus into the tympanic cavity. This is a diagnostic point of some importance as in my limited experience I have always found pus in the antrum when there was repeated attacks of paroxysmal pain that were relieved by a free flow of pus through a large perforation in the drum membrane.

The eburnating process had not advanced so much in the lower two-thirds of the mastoid but the cells of this region were small and filled with cholesteatoma, constituting a cholesteatomatous infiltration of the mastoid—a rare condition in my experience as this is the second case that I have met with in my practice.

There was a small perforation in the inner table overlying the lower portion of the sigmoid sinus, from which pus was bubbling; perforation was enlarged and a dram of pus liberated. The sinus wall was beginning to soften, and as palpation did not afford us any information, the outer wall was incised between two pressure pads and a fair sized parietal thrombus was taken out of the sinus.

This patient had a slow and tedious recovery, due to the lighting up of an old nephritis.

This case very forcibly illustrates the fact that we may have an infected thrombus, in one of the sinus of the dura mater, and yet there may not be manifested the symptom complex that we usually expect to see in these cases.

CASE 5.—This case came under my observation in 1904, at the instance of Dr. E. W. Hill.

History.—White male, age 23 years. Frequent attacks of earache in the left ear, but there has never been a noticeable fluid discharge from the ear, although he has frequently taken greenish crusts of a pungent odor from this ear. Present trouble began three weeks ago during an attack of tonsillitis, but mastoid swelling did not develop until two weeks later, that is one week previous to his first visit to my office.

Present Condition.—Temperature, 98° F.; pulse, 96. Pain deep down in the ear; edema limited to the lower two-thirds of the mastoid. Slight pressure over tip and antrum caused intense pain. Ear was freed of inspissated pus and cerumen. Inner end of membranous canal was found to be sagging and boggy. There was a large sequestrum of bone in the fundus of the canal, which could not be delivered through the external meatus. Mastoid does not transilluminate.

Saw patient at 3 p. m. and his temperature was 100° F., pulse, 100. He has had a number of attacks of vertigo and several rigors since his morning visit. The hearing power, for both high and low tones, is very much impaired. Advised operation at once, but the young man was under legal age and the consent of his parents could not be obtained before the following day.

June 10th.—Temperature, 98.8° F.; pulse, 98. Chill during the night which was followed by free perspiration. The ear is discharging a thin blood streaked material, from which a smear was taken, and the streptococcus was found to be the predenominating organism.

Four p. m.—Temperature 100.5° F.; pulse, 115. Marked increase in the objective symptoms. The father tells me that the boy had an acute ear condition in early infancy and that since that time there had been an accumulation of greenish malodorous crusts about the external meatus.

June 11th.—Operation; chloroform; subcortical cells and a large tip cell contained pus; antrum and tympanic cavity were filled with pus and cholesteatoma. The antral walls were undergoing dry caries as the bone came away under the curette like grains of sand.

A sequestrum comprising the inner end of the posterior and inferior walls of the external auditory canal was taken out, thus leaving the facial nerve exposed in its vertical portion. The inferior and anterior walls of the tympanic cavity were absent and the jugular bulb, the internal carotid artery, and the capsular ligament of the glenoid fossa were exposed. The wound was packed with strips of iodoform gauze and left open.

The young man was relieved of his pain and vertigo by the operation, but the fluctuations of temperature and pulse, and the slight chills were repeated three and four times a day; each chill was followed by a rise of temperature and pulse and mild perspiration. This condition continued for ten days before the consent of the parents could be obtained for another operation. During this ten days the temperature varied from 97° F. to 101° F. and the pulse from 110 to 140.

As the mastoid wound was in good condition, and the jugular bulb previous to the operation had been in gross communication with the pyogenic material in the tympanic cavity, it was thought that we had to deal with a primary thrombosis of the jugular bulb, and that small particles of the infected clot were becoming detached into the general circulation and thereby causing the form of bacteremia that was manifested.

On the tenth day following the primary operation the patient was anesthetized and the sigmoid sinus uncovered down to the bulb. A palpable clot was not discoverable, therefore, an aspirating needle of large caliber was inserted into the sinus and a free flow of blood followed but, when pressure was applied to the sinus above the needle, the flow immediately ceased. Pressure was maintained and the portion of the sinus was laid open with a small knife; a curette was introduced, and particles of softened clot were removed but we were unable to re-establish the circulation. It was deemed best to resect the jugular before resorting to further manipulations to free the sinus of the thrombus.

After the vein was excised, the sinus was incised down to the bulb and a great deal of disintegrated clot was taken out.

A gauze drain was inserted upward into the bulb, in order that it might bring away any softened material that had escaped our efforts. The temperature and pulse became normal within four days after the sinus operation and remained so throughout his convalescence.

This case serves to illustrate the difference between an actual involvement of the labyrinth by the pyogenic process and a labyrinth disturbance due to pressure transmitted through the fenestra in the inner tympanic wall, as the labyrinthine symptoms disappeared as soon as the pressure was removed by operation.

The low form of bacteremia that was manifested in this case may be accounted for in several ways: (1) That the clot in the bulb and lower part of the sigmoid sinus was disintegrating slowly, and thus such small infected emboli were detached into the blood stream that the phagocytes were able to perform their function as scavengers before the extreme constitutional disturbances, that are usually seen in a bacteremia, could be manifested. (2) It is quite possible that here may have been an attenuation of the organism responsible for the infection in consequence of the long continued course of the suppuration, just as we see it in chronic gonorrhea. (3) The individual equation of susceptibility and immunity or resistance to infection must be considered in a given case as the same conditions of infection in an individual of good resisting powers would not produce the violent train of symptoms that would be manifested in an individual that was greatly debilitated before the infection.

The wound in the neck healed by primary intention, and this, I think, was due to the method of technic that was followed in the resection of the vein. The technic of the procedure was as follows: A heavy pad of gauze was passed under the vein, preliminary to cutting it between the double ligatures, in order to prevent septic material in the severed ends of the vein from infecting the wound in the neck.

Before the stumps of the severed vein were dropped into the neck, their intimas were cauterized with carbolic acid and alcohol, just as the general surgeon does the stump of the severed appendix before he returns it to the abdominal cavity.

I have followed the above technic in several jugular resections, with the most gratifying results, as to primary union of the cervical wound, in cases that were manifesting a high degree of bacteremia.

Case 6.—White male, age 16 years. I saw this case in consultation with Dr. W. H. Metcalf, at 2:30 p. m., September 5, 1907.

History.—Discharge from left ear since early childhood as sequel of scarlet fever. There had been pain back of the ear on numerous occasions, when the discharge from the ear would cease temporarily.

Present trouble began about ten days ago during an attack of tonsillitis. He has had a number of mild chills and his temperature has fluctuated between 100° F. and 102° F. He had a very hard chill this morning, but his temperature was not taken until 1 p. m. when it registered 101° F.

Present Condition.—Temperature, 102.6° F.; pulse, 130; respiration, 22. He has an anxious drawn facial expression, that is suggestive of fear; skin is of a muddy yellow color; tongue is heavily coated; breath is foul; speech is slow and voice tremulous; there is an annoying dry cough but ascultation and percussion are negative. Has constant pain over entire left side of the head but is most severe in the mastoid region; he is extremely sensitive to pressure lightly applied over the tip, antrum, emissary vein and along the course of the internal jugular vein. There is edema, beginning about two inches above the external auditory meatus and extending downward to the clavicle. This edema is particularly noticeable in the occipital triangle, and there is exquisite tenderness to pressure applied in this region.

External canal is full of thick foul pus; drum membrane, malleus and incus are missing, and the inner tympanic wall is covered with polypoid granulations. The streptococcus was found to be the predominating organism.

There is a slight Babinski reflex and Kernig's sign is fairly well marked, as the angle of extension is about 120°; vision is indistinct and ophthalmoscopic examination revealed a double optic neuritis. The patient readily recognizes articles handed him and understands the purposes for which they should be used, but he displays considerable difficulty in naming the articles submitted to him.

There is a stable mydriasis of the left pupil and the right one responds very sluggishly.

Diagnosis.—Mastoid empyema, sinus thrombosis, and a provisional diagnosis of meningitis with brain pressure due to an extradural or subdural pus collection.

Admitted to hospital at 4 p. m.; temperature, 101.6° F.; pulse, 116; chill at 6 p. m., lasting 15 minutes; 7 p. m. temperature, 105.5° F.; pulse, 140; respiration, 28.

Operation began at 10 p. m. and the temperature was 102° F. The usual mastoid incision was later supplemented by posterior incision extending to the inion. Opening the cortex set free a thick, dark offensive fluid that was under considerable pressure as numerous bubbles could be seen in the fluid as it gushed out; large antrum was filled with cholesteatoma; inner table overlying the vertical limb of the sinus was completely eroded and the outer wall of the sinus was covered with granulations from which pus was exuding; granulations were removed and a perforation was found in the wall of the sinus; enlarged perforation and a disintegrated clot were mopped out of the sinus; ulceration of visceral wall of the sinus at the knee; ulcerated part of the sinus wall was cut out with small scissors and about three drams of pus was evacuated, exposing an area of superficial cerebral necrosis at this point. This is about the region of the naming center as pointed out by Mills, and possibly accounts for the patient's hesitation in naming objects.

On account of the extensive softening of the clot in the sinus, it was thought best to ligate and excise the internal jugular vein before attempting to free the sinus of the disintegrated thrombus. The jugular was found to be thrombosed to one-half inch below the facial vein, the latter vein also contained a clot. The jugular was excised below the lower limit of the clot and the stump of the vessel was stitched into the upper angle of the cervical incision.

Sinus had to be uncovered to the torcular before the posterior axis of the softened thrombus could be reached. This sinus was next curetted in the direction of the bulb but a return circulation was not established. A stream of normal saline solution was introduced into the sigmoid sinus in the direction of the jugular bulb and particles of softened clot washed out of the end of the jugular that was stitched into the neck wound. The superior petrosal sinus was opened inward as far as the internal auditory meatus without establishing a return flow.

The posterior osseous canal wall was taken down and the middle ear freed of granulations. Posterior horizontal incision closed and wound packed with gauze. Lower two-thirds of neck wound closed but it suppurated and had to be reopened.

September 6th, 6 p. m.—Temperature, 99.4° F.; pulse, 84. Chill at 6:30 p. m., chill at 10:30 p. m., following which the temperature was 104.2° F. and pulse 130.

September 7th.—Chill at 8:30 a. m., followed by a rise of temperature to 104° F. and pulse 140. During the night he complained of pain over the upper portion of the left lung; the respirations are shallow and increased in frequency; large moist rales heard on auscultation and there is prune juice expectoration; has developed a diarrhea and the stools are extremely offensive.

Dressing changed and the entire wound found covered with pyogenic membrane; all stitches taken out and wound swabbed with turpentine; hot compresses of permanganate of potash solution ordered which were to be changed every twenty minutes. Chill at 7:30 p. m.; one hour afterward the temperature was 103 6° F., pulse 160.

September 8th, 7 a. m.—Temperature, 99.5° F.; pulse, 96. Chill of thirty minutes' duration and the temperature soon registered 104.5° and pulse 126. Slight edema and discoloration of the lids of the left eye.

September 11th.—For the past forty-eight hours the temperature has fluctuated between 98° F and 102° F., and the pulse has varied from 96 to 116. Patient has been exceedingly restless and at times wildly delirious; Kernig's and Babinski's signs are positive and there is some retraction of the head; edema and discoloration of the lids of the left eye have increased.

September 12th.—Exophthalmos of the left eye; edema of lids of right eye; projectile vomiting several times during the day; unconscious at 9 p. m. and died six hours later.

Autopsy not permitted.

This patient was given 140 cc. of streptolytic serum and twelve hypodermic injections of Declat's preparation of phenic acid besides the usual remedies that are in vogue in the treatment of pneumonia.

This case as well as the preceding one serves to illustrate the fact that in an acute exacerbation of a chronic purulent otitis media in which there are symptoms of mastoiditis, we should resort to radical operative interference at once if we wish to conserve the interests of our patient. They also serve to show us the extensive destruction that may take place in the temporal bone without the manifestation of subjective symptoms. From the findings at the operations upon these patients I am of the opinion that chronic suppuration of bone had been in progress for months, if not years, as it is hardly conceivable that such destruction of osseous tissue could be accomplished in one or two weeks. Every surgeon who has had a wide experience in dealing with chronic suppuration of bone, has often been surprised at the mildness of the symptoms and the advanced pathologic conditions that he finds at the time of operation.

On the day following the operation, the young man appeared to be better, inasmuch as the pain was relieved, and he was able to name objects readily, the later condition would indicate that the naming center had not been destroyed, but as has been suggested by Mr. Ballance, the connecting fibres between this center and the speech center were obtunded to a certain extent by pressure from the subdural abscess.

Cases, like the one under discussion, which manifest symptoms of meningitis before operation, do not offer a very favorable prognosis, and in this case the Babinski and the Kernig signs were positive, and we were fully warranted in assuming that we had a complicating meningitis.

As this patient had an annoying cough previous to the operation, although auscultation and percussion were negative, I cannot but conclude that a metastatic pneumonia was developing at that time, as clinicians of experience in diagnosticating diseases of the lungs tell us that it is often impossible to make a diagnosis of central pneumonia by auscultation and percussion, and that they have to wait for the appearance of the characteristic expectoration.

I am well aware that, during the manipulations that are necessary to clear a thrombosed sinus, one may dislodge a portion of the infected clot into the general circulation, and that it may be carried by the blood stream to the right heart, and thence to the lungs, to form the focus for a metastatic pneumonia, but I cannot concede that this occurred in the case before us, as the clot in the bulbar end of the sinus was not disturbed until after the internal jugular had been excised, and pressure was made on the opposite jugular while we were clearing the torcular end of the sinus.

The foul smelling purulent stools that the boy had would indicate that his diarrhea was due to a septic enteritis and not to an excess of carbon dioxid in the blood which was the cause of the frequent loose stools in the late stage of pneumonia.

The clinical symptoms of exophthalmos, edema and discoloration of the lids of the left eye, and the later manifestation of the same symptom in the right eye in connection with the fact that we were unable to get a return flow from the superior petrosal sinus, would amply justify us in concluding that there was a septic thrombophlebitis of both cavernous sinuses; this being the case it is quite easy for us to see how the infecting element could be carried to the lungs through the right internal jugular.

It is in cases like this one that the diagnosis of a complicating brain abscess is rendered extremely difficult, if not impossible, as the symptoms of pyemia so predominate that they obscure the characteristic signs of low temperature and pulse that are seen in abscess of the brain.

XVI.

DIAGNOSIS AND TREATMENT OF SUPPURATION OF THE LABYRINTH.

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The diagnosis of suppuration of the labyrinth is made from the symptoms evinced by the patient before operation and by the pathologic condition discovered during operation.

Symptoms.—The invasion of the labyrinth by a suppurative process in the middle ear is marked by no symptoms in the majority of cases. This is due to the fact that it occurs generally during a chronic suppurative otitis media associated with caries or cholesteatoma. Extension takes place slowly by a process of erosion from without. This allows the labyrinth apparatus to accommodate itself to the gradual change in pressure and condition that occurs with the localized destruction of its bony capsule. It is the sudden invasion of the labyrinth or the sudden extension of disease within a labyrinth already entered that gives rise to symptoms. When this occurs it causes certain manifestations that are of diagnostic value.

Of these deafness is of importance. Unilateral deafness, as Bezold has pointed out, is difficult of diagnosis and consequently of doubtful value. When, however, a patient suffering from a suppurative otitis media develops under observation a sudden increase in deafness in the suspected ear, the symptom is of great significance. The absence of deafness does not necessarily mean that extensive pathologic change has not already taken place in the labyrinth for the vestibule may be filled with cholesteatoma and pus without materially affecting the hearing. Even the cochlea may be extensively diseased without destroying hearing for conversation.

Disturbances of equilibrium are frequently present. When standing the patient often rests most of his weight upon the foot of the affected side. While walking he tends to turn off or bend to the affected side.

These disturbances are intensified if the eyes are closed. In some cases they are slight but continuous while in others they alternate with periods during which the patient is free of all inconvenience. In severe cases they are so marked as to prevent the patients from assuming the upright position or moving about without support. After being present for some length of time they have been known to disappear for years.

Vertigo is common and is intensified by exertion. It varies greatly in degree and character. In some cases it is not more than dizziness, transient or continuous. In others typical and severe rotary vertigo appears. In one of my cases pressure upon the right semicircular canal after operation caused all objects to rotate very rapidly to the right. Removal of pressure causes them all to snap back through the reverse arc quickly until they reached their proper position. Pressure upon the posterior semicircular canal caused the bed to slip suddenly away toward the foot. Removal of pressure brought it quickly back again. These sensations were so intense that that patient would hold tightly to the bed to keep from throwing himself on to the floor.

In cases in which vertigo is most marked nausea and vomiting are generally noted. They occur upon arising or follow exertion.

Lateral nystagmus is less common. It rarely occurs without vertigo and is most marked when the eyes are turned toward the healthy side.

Fever and headache are rather uncommon and are not at all characteristic.

The hearing test is of questionable value. The hearing for the voice in my own cases varied from the whisper at three feet, to a degree which made it probable that the loud conversational voice was heard in the other ear although that ear was hermetically sealed. The fact that a very high degree of deafness often occurs in chronic suppurative otitis media without extension of the infection to the labyrinth and that extension into the semicircular canal system sometimes takes place without causing marked deafness, lowers the value of the voice test considerably even when taken into consideration with other tests and renders it worthless when considered alone.

The Weber is decidedly unreliable. It was not localized in several of my cases and in others was localized in the suspected ear quite as frequently as in the other.

The Rinne was negative more often than it was positive.

The high limit, however, was always somewhat lowered, in one case was 512. In this case the Weber was not localized. The lower limit is of course elevated on account of the middle ear involvement.

The symptoms mentioned are important in that they point toward the presence of an intracranial complication but are not sufficient in themselves to render a diagnosis of labyrinth disease positive. They may all be present in cases in which the labyrinth is not involved. This is demonstrated by the following case:

Mrs. S., 30 years. Scarlet fever at the age of 4 years. Both ears discharged at that time. Right ear soon ceased to discharge but left has continued to discharge at times ever since. At time of first discharge had complete left facial paralysis. This lasted for about two months. Since then has had times when paralysis was apparent lasting several weeks. Since first attack has never been complete. About two weeks ago during a head cold was taken with severe rotary vertigo, ringing tinnitus and increase in deafness in the left ear, pain in the left occipital region, slight foul discharge from left ear. Temperature has reached as high as 105 with frequent remissions; no chills, yesterday delirium, pupils dilated, breathing difficult, apparently in extremis. Today remarkable change for the better.

Examination.—Patient well developed, intelligent woman, perfectly rational, but inattentive, complaining of pain in region of left ear, high pitched tinnitus left ear, temperature 101, pulse 80, respiration 20, leucocytosis 14,000. Knee jerks slightly exaggerated. Right ear shows evidence of old suppuration but no sign of acute inflammation. Left ear, membrana tympani present, retracted, scar in superior half, small perforation in postero-superior quadrant, contains somewhat tenacious pus, probe passes into attic. No drooping of posterior superior canal wall, no mastoid tenderness. Hearing test somewhat unsatisfactory on account of patient's condition.

Right.		Left.
Loud conversation	Voice.	Loud conversation
Not localized	Weber.	Not localized
Negative	Rinne.	Negative
256 slightly higher forks 0	A. C.	256 slightly higher forks 0
Apparently somewhat decreased	B. C.	Apparently somewhat decreased.
Dench 3.	High.	Dench 4
256.	Low.	256

Paracentesis permitted escape of foul pus and cholesteatoma. Diagnosis of chronic suppurative otitis media, cholesteatoma and probable involvement of the labyrinth was made and radical mastoid operation advised with exploration of labyrinth if indicated at time of operation. Operation was refused. Patient remained in same condition for two days and then had another attack of pain.

Rise of temperature to 105 without chill, increase in deafness, tinnitus and vertigo from which she made a slow recovery. Ten months later a radical mastoid operation was performed. Tympanum and mastoid were found filled with cholesteatoma but the labyrinth was intact. Complete recovery. It is interesting to note that after the radical operation the upper limit became normal and hearing for conversational voice increased to six feet.

The labyrinth symptoms and hearing test indicative of labyrinth involvement were no doubt due to pressure upon the labyrinth of the cholesteatoma and to incident congestion.

Even more important than the fact that symptoms indicative of invasion of the labyrinth may be present during the course of a chronic suppurative otitis media without disease of the labyrinth is the fact that invasion of the labyrinth may take place without the presence of a single symptom on account of which the condition may be suspected. Of my own cases marked deafness was present in three, disturbances of equilibrium in five, vertigo in five, nausea and vomiting in three, nystagmus in two, no symptoms whatever in five.

The diagnosis of labyrinth suppuration can at present be made with certainty, in the great majority of cases at least, only at the time of the radical mastoid operation, by discovery of disease of the inner tympanic or mastoid wall and of a sinus leading into the interior of the labyrinth. These sinuses appear in order of their frequency in the semicircular canals, through the oval window, through the promontory, and through the round window. Perforations into the horizontal semicircular canal is frequently easy of diagnosis. This is true when the perforation occupies a point just below the most prominent portion of the semicircular canal. In such cases it appears as a single opening into the lumen of the canal, or if more destruction has taken place as two openings one leading into each branch of the canal. (I have seen several such perforations without signs of caries, granulation tissue or pus within the canal.) The diagnosis is more difficult although the destruction is greater in cases in which the posterior half of the horizontal semicircular canal is lacking. With the perforation in this position, the operator may be left in doubt as to its existence despite perfect illumination and painstaking investigation. Pus is rarely seen issuing from such a perforation.

I have seen it in but one case. In that case the vestibule was filled with fluid pus. More frequently a small bud of granulation tissue is noted within the canal. When this is wiped away the observer may be in doubt as to whether the interior of the canal is involved or not, possibly concluding that the granulation tissue is due entirely to the disease within the antrum. I believe that such indecision is justified and that perforation may occur in the semicircular canal, exist for a time, become closed by granulation tissue and later by bone with but slight, if any, involvement of the labyrinth. In one case, in shaving down some cancellous bone over the horizontal semicircular canal in a patient in whom no labyrinth symptoms had been noted I cut away a small irregular mass of bone, the removal of which opened the canal. This I think was new bone formed in the successful attempt on the part of nature to close a perforation and protect a labyrinth from invasion.

The posterior semicircular canal is somewhat more deeply situated than the horizontal and is more deeply covered with cancellous bone consequently its exact delineation is somewhat more difficult. On this account perforation through the canal is sometimes missed. When the canal is thoroughly exposed the perforation is easily seen occupying the most prominent portion of the canal. It is sometimes seen on the anterior surface and associated with destruction of the posterior branch of the horizontal canal. In some cases very small pneumatic cells may cover the canal and be especially numerous in the neighborhood of the solid angle. They sometimes extend rather deeply along the walls of the canals and if filled with freely bleeding granulation may be mistaken for a perforation. In this connection the systematic use of the fine probe is of great diagnostic value. This instrument must be employed with judgment. It is well recognized that in cases which exhibit a single perforation in this neighborhood the labyrinth disease is frequently definitely localized and that this localizing process takes place by the formation of a wall of protecting granulation at the deepest limit of the disease. It is without question highly dangerous to pass a probe further into the semicircular canal than this protecting wall. Such a procedure only carries infection into the deeper portions of the labyrinth and disseminates the disease. It does not afford the observer any additional information. As the point at which this protecting wall has formed can not be told, the probe is to be passed only deep enough to render the diagnosis of perforation positive.

It is generally stated that next in frequency to the neighborhood of the semicircular canals perforation takes place through the oval window. In the presence of the stapes this perforation must be difficult of diagnosis. In the absence of the stapes, perforation can sometimes be made out if not by inspection, then by means of the fine probe. Hinsberg states that infection can pass through the oval window in cases in which macroscopic change in the window cannot be discovered.

In support of this statement I have been able to demonstrate caries of the vestibule in a case in which I could not discover a perforation. The absence of the stapes is not always significant of perforation through the oval window as it was noted in one of my cases in which no perforation could be detected and in which fair conversational voice was present after operation. I have seen perforation of the promontory in one case in which about two-thirds of the promontory was lacking. Cases have been reported in which infection reached the labyrinth from the cranial cavity through the internal auditory meatus or the aqueducts. I am inclined to think that the acqueductus vestibuli was the avenue of infection in one of my cases.

Much difference of opinion exists concerning the pathologic significance of perforations in various positions in the external labyrinthine wall.

In my cases, judging from hearing test and result of operation, single perforations into the horizontal semicircular canal occurred only in cases of circumscribed labyrinthine disease. Double perforations, one in the horizontal and one in the posterior semicircular canal, occurred in two cases in which the vestibule was filled with pus and cholesteatoma. It would seem that double or multiple perforations must always speak for an extensive pathologic condition. In considering the value of visable perforations in the diagnosis of labyrinth suppuration secondary to suppurative otitis media it must be remembered that the disease has been demonstrated clinically and at autopsy in cases in which perforation could not be recognized.

The indications for operation upon the labyrinth are met in two classes of cases:

1. Those in which symptoms referable to labyrinth disease have not been present before operation.

(a) When a single perforation is discovered through a semicircular canal, from which pus escapes.

(b) When a perforation through the oval window is discovered, whether pus is seen or not. In these cases marked deafness is present and extensive disease of the vestibule must be suspected.

(c) When labyrinth symptoms develop suddenly after a radical mastoid operation. The majority of these cases are due to a latent labyrinth suppuration, an acute exacerbation of which has been set up by the operation. In such cases the vestibule is to be opened widely and further operative measures governed by its condition. In isolated perforations through the horizontal semicircular canal in which no other evidence of disease can be demonstrated operation upon the labyrinth may be postponed.

This is in support of Hinsberg's statement that in his experience, of all cases of circumscribed semicircular canal disease not a single patient succumbed to the consequences of the labyrinth suppuration. In this connection he points out the necessity for watching, very carefully, cases in which a single perforation of a semicircular canal has been demonstrated so that on the appearance of vertigo, nystagmus and diminution of hearing, the labyrinth may be promptly opened.

2. Those in which labyrinth symptoms have been present before operation:

(a) When no fistula is found.

(b) When fistula is found.

When no fistula is found. This class of cases, it seems to me, should be very small and should include those cases which have developed labyrinth symptoms while under observation and those in which deafness typical of disease of the perceptive apparatus can be demonstrated. Symptoms highly suggestive of labyrinth suppuration so frequently disappear after the radical mastoid operation that unless the pathologic change in the tympanum and mastoid is altogether too slight to account for the labyrinth symptoms delay may be allowed. To this class belong also those cases in which a deep seated cerebellar abscess or extradural abscess is suspected (Jansen).

When a perforation into the labyrinth is found in a case that has previously shown labyrinth symptoms the indication for opening the labyrinth is clear.

The following six cases are illustrative of the fact that invasion of the labyrinth can take place without symptoms, while the next five show some symptoms characteristic of the condition.

August 17, 1905. CASE 1.—Percy B., 15 years. Discharge from left ear following scarlet fever eleven years ago. Discharge has persisted with infrequent remissions ever since. No symptoms except moderate deafness and foul discharge.

Examination.—Right ear negative. Left, great destruction of M. T. Tympanum filled with granulation, pus and cholesteatoma. No mastoid symptoms. Large amount of adenoids.

Right.	Hearing Test.	Left.
Loud conversational voice	Voice	20/21
+	Weber	
—	Rinne	+
Dench 3	High	N.
512	Low	N.

Radical mastoid operation discovered tympanum filled with cholesteatoma, antrum containing moderate amount of granulation tissue, pus and cholesteatoma, malleus and incus wanting. Small sinus leading into posterior branch of horizontal semicircular canal. No evidence of pus in canal, no caries. Koerner flap and skin grafts later. Sinus moderately enlarged for purpose of examination. Adenoid operation. Slight vertigo after operation.

May 3, 1906. Case 2.—Ivan P., age 10 years. Chronic suppurative otitis media since scarlet fever seven years ago. No symptoms except moderate deafness and discharge.

Examination.—Right ear. Great destruction of membrana tympani. Ossicles not seen. Tympanum contains granulation, pus and cholesteatoma. Probe passes into attic and discovers caries on external attic wall and in direction of antrum. Left ear: Scars in membrana tympani. No evidence of present suppuration.

Right.	Hearing Test.	Left.
L. C. V. at ear	Voice	15/21
+	Weber	
—	Rinne	+
Dench 2	High	N.
512	Low	256

Right radical mastoid operation. Cholesteatoma in tympanum, antrum and perisinus collection of cholesteatoma. Perforation in horizontal semicircular canal leads into vestibule. This opening enlarged. Thiersch grafts in Eustachian tube and tympanum. Koerner flap. Initial incision sutured. Recovery without marked labyrinth symptoms.

July 8, 1907. Case 3.—Fred H., age 17 years. Discharge from right ear for fifteen years without symptoms.

Examination.—Right ear: Polyp occludes canal, from around polyp, escapes foul cholesteatoma. Sinus through posterior canal wall passes into mastoid.

Right.	Hearing Test.	Left.
Loud conv. voice	Voice	20/21
+	Weber	
—	Rinne	+
Dench 2	High	N.
512	Low	N.

Radical mastoid operation. Tympanum, antrum and mastoid filled with true cholesteatoma. Dura over middle cerebral fossa and over sinus laid bare by the disease. Polyp attached to inner wall of antrum. Sinus through horizontal semicircular canal leads into vestibule. Sinus enlarged. Koerner flap, initial incision sutured. Moderate vertigo for several days after operation. Discharged well.

November 21, 1907. CASE 4.—Madge S., age 8 years. Recurrent discharge for the past three years, continuous from the left ear for the past year. No history of labyrinth symptoms.

Examination.—Membrana tympani shows great destruction. Tympanum contains cholesteatoma and pus. Ossicles not visible. No mastoid tenderness. After several weeks' treatment without avail, radical mastoid operation. Most of the disease in and in the vicinity of the antrum, which contained cholesteatoma and caries. Fallopian canal lacking for about one-fourth inch in the immediate vicinity of the horizontal semicircular canal. Horizontal semicircular canal shows almost complete destruction; remainder of canal carious. Dura and sinus laid bare. Short Koerner flap. Initial incision sutured. Skin graft later. Complete recovery without labyrinth symptoms.

December 30, 1905. CASE 5.—Frances McD., age 18 years. Continuous discharge from both ears since measles thirteen years ago. Severe headache but no other symptoms.

Examination.—Both ears showed marked destruction of membrana tympani. Tympanum contains cholesteatoma and pus. Caries in both attics. Left shows considerable destruction of posterior canal wall.

Right.	Hearing Test.	Left.
8/35	Voice	5/35
Dench 2	Weber	+
—	Rinne	—
	High	Dench 2
512	Low	512

January 11, 1906.—Radical mastoid operation left side. Extensive sclerosis. All of posterior and posterior half of horizontal semicircular canal found destroyed. Opening enlarged. Koerner flap. Thiersch grafts to tube and tympanum. Moderate vertigo after operation.

Stacke operation on the right side at a later date. Labyrinth not involved.

August 28, 1905. CASE 6.—Cleo F., age 15 years. Both ears have discharged since the age of 6 months. No nausea; no dizziness or other labyrinth symptoms.

Examination.—Both ears show canals filled with granulations, pus and cholesteatoma. Sinus leads through the posterior canal wall of the left ear into mastoid. Adenoids and tonsils.

Right.	Hearing Test.	Left.
L. C. V.	Voice	L. C. V.
Dench 3	Weber	+
—	Rinne	—
	High	Dench 3
1024	Low	1024

Radical mastoid operations on both ears at different dates. Right side showed moderate amount of cholesteatoma, granulation tissue, and pus throughout tympanum and mastoid. Labyrinth had been invaded through the posterior semicircular canal which had been completely destroyed. Horizontal semicircular canal also involved. Thiersch graft of tympanum, initial incision sutured. Moderate labyrinth symptoms after operation. Mastoid operation followed by removal of adenoid.

Left radical mastoid operation showed extensive cholesteatoma formation with exposure of the facial nerve but no involvement of the labyrinth.

March 15, 1907. CASE 7.—Frank R., age 14 years. Right ear has discharged for ten years, cause unknown. No symptoms until one month ago when complete facial paralysis and vertigo with increase in deafness. Patient says that at that time and for several days, he staggered to right but did not fall.

Examination.—Canal occluded in its middle third by drooping of superior canal wall which touches inferior. By inserting speculum beyond occluded area small amount of stale discharge and glistening polyp are seen. Complete facial paralysis. No mastoid tenderness. Adenoids and tonsils.

Radical mastoid operation. Tympanum, antrum and mastoid filled with foul pus. Fallopian canal shows perforation about one-fourth inch long, downward from level of horizontal semicircular canal which shows perforation, which uncaps canal and shows openings into both branches. Openings enlarged. Koerner flap. Initial incision sutured. Insignificant vertigo after operation. Adenoids and tonsil operation at a later date. Complete recovery from facial paralysis in three months.

May 13, 1906. CASE 8.—Wm. H., age 22 years. Right ear has discharged since measles seventeen years ago with occasional exacerbations. Polyps removed from both ears one year ago. Two months go while undressing had his first attack of vertigo which was so severe that he fell over on to the bed. This vertigo has continued with periods of remission and exacerbation up to the present time. No nausea or vomiting. Acknowledges syphilis several years ago. No recent symptoms.

Examination.—Patient sits with head bent to right. Not interested in surroundings. Answers questions slowly but correctly. Apparently not in pain. Pupils equal, react normally, no nystagmus, no strabmisus. Disturbance in equilibrium marked upon arising, stands with most of his weight upon the right foot, while walking turns off and bends to the right.

Ears: Right, canal contains creamy pus. Depths of canal filled with easily bleeding granulations. Drooping of superior wall of the canal just external to the tympanum. No mastoid tenderness. Left, dry depths are filled with bloody crusts.

Right.	Hearing Test.	Left.
L. C. V.	Voice	2/35
Not localized	Weber	Not localized
—	Rinne	—
512	High	N.
512	Low	512

Radical mastoid operation right side. Marked superficial sclerosis of mastoid. Deeper parts filled with cholesteatoma. Horizontal semicircular canal completely destroyed. Granulations in tympanum. Koerner flap, sewed to posterior wall of concha. Initial incision not sutured. Riverdan skin grafts at a later date. For several days severe rotary vertigo to the right. This gradually wore off. While walking the patient had the inclination to turn off to the right but was able to walk in a straight line with some effort. Three months after operation had two attacks of severe vertigo with some nausea. Examination revealed complete epidermitization of the operative field. No apparent trouble. Since then no report of patient.

August 5, 1905. CASE 9.—Patty Van T., age 9 years. Has had continued discharge from both ears since scarlet fever six years ago. Two weeks ago severe pain right side, with decrease in amount of discharge, postaural swelling on the third day. Since attack patient has been very dizzy but has been too sick to complain.

Examination.—Patient evidently very ill. Temperature 103, pulse 110, very weak and thready, sepsis, adenoids.

Right ear, canal occluded by drooping of superior wall, large postaural swelling extending to cheek. Cholesteatoma in canal.

Left ear, membrana tympani gone, tympanum filled with cholesteatoma. Right radical operation with curettage of vestibule. Cortex, paper thin. Five perforations externally. Tympanum, antrum and mastoid filled with cholesteatoma. Two perforations in middle cerebral fossa. Pus and cholesteatoma on sigmoid sinus. Posterior semicircular canal and posterior half of horizontal semicircular canal destroyed. Vestibule filled with pus. Left radical mastoid at later day. Labyrinth not involved. Discharged well.

October 8, 1906. Mrs. Wm. J. B., age 42 years. Patient had scarlet fever at age of 5 years. Both ears discharged for several years thereafter. Later the right ceased but the left has troubled her until the present time, slight facial paralysis. For about a year she has had frequent and severe sick headaches, with nausea, vomiting and rotary vertigo. Three weeks ago during a severe headache she suddenly became unconscious. This lasted for several minutes after which she was helped to bed. Since then she has had continuous headaches over the entire head. Patient is two months pregnant but thinks that that has nothing to do with her symptoms, judging from previous experiences.

Examination.—Right ear shows great destruction of membrana tympani. Tympanum dry. Left ear, two perforations in posterior half. Moderate amount of discharge having the odor of cholesteatoma.

Right.	Hearing Test.	Left.
L. C. V	Voice	L. C. V.
	Weber	+
N.	Rinne	0
512	High	15 D.
	Low	—

Patient is very unsteady on her feet, sways from right to left and has some difficulty in maintaining the upright position. When walking turns off the to left unless she is careful.

Radical mastoid operation. Mastoid completely sclerosed. No antrum discovered. The position of the antrum could be definitely made out but the cavity itself was filled with new bone. Tympanum filled with cholesteatoma. Promontory lacking in great portion, this causing a perforation in vestibule and first whorl of cochlea. This opening enlarged. Following operation vertigo was marked for ten days after which it gradually disappeared. Discharged well.

CASE 11.—Tiny B., school girl, age 14 years. Was brought to the University Hospital on account of a suppurative middle ear disease of the left side.

History.—Patient had scarlet fever at the age of 1 year. Both ears discharged at that time. At the same time a swelling appeared behind the ear. This was lanced and the wound soon healed. The discharge from the ear, however, persisted for about two years. From that time a little discharge appeared at times. About a year ago during a head cold pain in the left ear reappeared together with an increase in the amount of discharge. Two weeks previous to admission to the hospital, during a head cold pain reappeared and discharge ceased. Two days previous to admission a swelling was seen over the mastoid. About the same time child became much weaker. She has had at no times chills, nausea or vomiting.

Examination.—Shows a child fairly well nourished lying upon her right side, head markedly retracted, legs flexed on thighs, face flushed, eyes closed, mouth open, patellar reflexes absent, Kernig's sign marked. The heart while fairly strong when patient is lying down becomes irregular and weak during exertion. She answers questions intelligently but slowly and sometimes requires several efforts before she is able to finish her sentence. She protrudes the tongue fully but with some hesitation and unsteadiness. Temperature 102.8, pulse 140, respiration 24, leucocytes 17,000.

Local examination.—Right ear shows effect of a previous suppuration. Left ear, external auditory canal filled with foul cholesteatoma and pus. Lumen of canal narrowed by pressure from mastoid. Over the lower half of the mastoid is present a smooth swelling, very sensitive to pressure and showing slight fluctuation. This swelling extends beneath mastoid tip into the deep neck muscles.

A diagnosis of chronic suppurative middle ear disease, Bezold's mastoiditis with cholesteatoma and meningitis was made and a bad prognosis given. Careful questioning of the mother showed that the child had begun to complain of headache only the day before and that all her bad symptoms had come on suddenly. Therefore it might be supposed that her meningitis had begun within the previous few hours, had come on suddenly, was progressing rapidly and might at that time be confined to an area that could be reached by operative measures. The writer could recall five cases of purulent otitis meningitis operated upon successfully in his own practice and therefore felt warranted in suggesting an operation as the only possible means to offer even the slightest chance of recovery. The child was therefore immediately prepared for operation.

Under ether anesthesia the usual mastoid incision was continued down over the area of swelling in the neck and several drams of foul pus and cholesteatoma were allowed to escape. Perforation through the inner surface of the mastoid tip had taken place on to the deep muscles of the neck. The mastoid cortex was completely removed and the mastoid cavity found of moderate size and filled with a true cholesteatomatous tumor having a well defined limiting membrane. This membrane had ruptured and allowed the escape of its fluid contents. Careful removal of the tumor showed the bony wall of the inner table destroyed by the disease so that the cerebellum was exposed over the area about the size of a silver dollar. Over this area, the membrane of the cholesteatoma was firmly adherent to the cerebellar tissue, i. e., a true subdural cholesteatoma. The dura in the neighborhood was part covered with foul purulent granulations. Those were removed and the brain uncovered until healthy dura was uncovered on all sides, except over a small area near the median line. The reason for not going further in this direction will be explained later. The lateral sinus was found uncovered for a distance of about an inch and a half but its lumen had not been invaded. The horizontal semicircular canal had been destroyed and the labyrinthine vestibule opened. A large amount of pus was secured from the posterior cerebral fossa. All pneumatic structure infected was then removed including the mastoid tip and the posterior root of the zygoma and the wall of the external canal, thus converting the middle ear, canal and mastoid into one cavity with smooth walls. The tympanic end of the Eustachian tube was then curetted in order to secure its future closure and a counter opening made in the neck for drainage. At this point, in spite of hypodermic stimulation and of hot saline enemata repeated during operation the patient suddenly showed signs of complete collapse. Further operating was postponed and the patient returned to bed where she made a slow recovery from the anesthetic, regaining consciousness eight hours later.

9 p. m.—Patient was perfectly conscious but restless. She answered questions satisfactorily but slowly. Protrusion of the tongue showed paralysis of the left half of the tip. Opisthotonos became more marked with double optic papillitis.

May 10, 1907.—She suddenly became comatose and died,

Autopsy.—Brain showed slight tenseness of dura, considerable congestion of the pia, slight milky exudate over the left temporal lobe. Several drams of plastic exudate over left cerebellar area corresponding to field of operation and extending inward and downward so as to involve structures of the base. True cholesteatoma membrane adherent to pia, left lateral ventricle filled with flaky fluid. Choroid plexus congested. Cord shows several drams of cloudy fluid in the arachnoid of the lower dorsal and lumbar cord. The fact that the cholesteatoma occupied a position in immediate relationship with the internal auditory meatus, and that the changes in the vestibule were much greater than those in the semicircular canal system makes the inference possible that infection reached the labyrinth through the internal auditory meatus, and that the perforation through the horizontal semicircular canal took place from within outward, at least examination of the bone makes it evident that the changes within the vestibule antedate those in the canal.

TREATMENT.

In all cases in which labyrinthine suppuration has been suspected before operation or has been diagnosticated at the time of operation by the discovery of a sinus leading into the labyrinth the radical operation must be performed. Not only is this the only operative procedure by which the suppurative process can be dealt with but it is the only one that gives a satisfactory view of that part of the labyrinth wall through which infection has penetrated.

It has already been pointed out that infection reaches the labyrinth through the region of the semicircular canal, the oval window, the round window or the promontory. In order thoroughly to expose these areas, the last steps in the radical operation must include the careful delineation of the three canals, the complete obliteration of the facial ridge even to the exposure of the facial nerve, the removal of the inner end of the anterior canal wall and of the external wall of the hypotympanum. The depths of the wound are then packed with gauze soaked in adrenalin in order that the field may be rendered bloodless and careful inspection of the labyrinth wall facilitated.

The complete exenteration of the labyrinth includes three distinct steps:

1. The entrance into the vestibule through the semicircular canal system.
2. The counter opening through the oval window.
3. The operation upon the cochlea through the promontory.

The operative field is so contracted, the relationship between the vital structures so intimate that a perfect orientation must be secured by work upon the cadaver before the exploration of the interior of the labyrinth can be attempted with safety to the patient or credit to the operator.

The entrance into the vestibule through the semicircular canal system is chosen as the first step in cases in which perforation through a canal can be demonstrated, as it offers a more comprehensive view of the vestibule than does the opening through the oval window. It is attended by less danger and less difficulty, it is less likely to affect the hearing disastrously and finally it sometimes reveals a localization of the pathologic process that renders further operative interference unnecessary. Removal of the semicircular canal system follows their satisfactory delineation. It is conveniently begun by shaving down with a curette or sharp chisel the superior margin of the horizontal semicircular canal in its posterior half.

This is continued until the canal is well opened. In the same way the posterior canal is widely opened. Then by means of the mallet and sharp narrow chisel the superior wall of the horizontal canal is followed directly inward until the vestibule is reached. The posterior and superior canals are then obliterated. The chief danger during this step in the operation lies in injury to the facial nerve. This is avoided by not interfering with the inferior wall of the horizontal canal which is in most intimate relationship with the Fallopian canal. I have demonstrated many times upon the cadaver that the chisel must under no circumstances be allowed to impinge upon the wall of the Fallopian canal, nor to rest upon the crest of the previously opened horizontal canal while this canal is being obliterated, as the Fallopian canal will certainly be fractured. It is a wise precaution, I think, against injury to the facial to uncover the nerve for a considerable area of the horizontal portion in order that its exact position may be determined. Care is to be used in curetting the inner wall of the vestibule which is very thin, and rupture of which is attended by considerable danger, to both facial and auditory nerves. If painstaking examination seems to show that the disease is limited to the semicircular canal system further operative measures may be postponed. If, on the other hand, the interior of the vestibule suggests further extension of the disease or if a sinus leading through the oval window or promontory has been discovered the second step is undertaken. This procedure may be carried out as the first step in the operation if no perforation through canal is found. This consists in enlarging the oval window downward by means of a very sharp narrow chisel. In this way the posterior half of the inner tympanic wall is removed. With the greatest caution in order to avoid fracture of the inner wall of the vestibule into the internal auditory meatus, this opening is enlarged until satisfactory examination of the vestibule can be made.

The third step is the operation upon the cochlea. It is carried out in the cases only in which disease of the cochlea can be demonstrated. This, as J. D. Richards has recently pointed out, is the most dangerous step in the operation not only on the account of the most intimate relationship that the carotid artery, the jugular bulb, and the facial nerve bear to the field of operation, but especially on account of the position the very delicate and fragile modiolus that occupies the center of the cochlea. The modiolus is a hollow cone whose side forms the peripheral end of the internal auditory canal and whose apex is at a point an eighth of an inch in front of and on a level with the oval window. From his experiments and observations Richards concludes that the apex of the cone can be removed down to the first cochlear whorl without bringing the operative field and the intradural sac into gross communication and so rendering a fatal meningitis probable. Measurements of a number of temporal bone seems to prove to me that the internal auditory meatus terminates between one-eighth and three-sixteenths of an inch internal to the promontory. Consequently to do more than uncap the pyramid must be exceedingly dangerous and is rarely necessary. The cochlea is opened safely by choosing a point one-fourth of an inch in front of the lower level of the oval window and chiseling downwards and forward very gently until a cochlear whorl appears. This is enlarged backward to the vestibule and forward as far as possible, chiseling as Richards advises always in the direction of the long axis of the pyramid. In this way two cochlear whorls are widely opened. This is followed by the cautious removal of the remainder of the promontory which permits inspection of the entire upper whorl, and almost all of the lower whorl. My experience upon the cadaver leads me to believe that in all cases in which indications are not absolutely plain, operative measures should cease at this point, although a small portion of the upper half lower whorl has not been seen. Even if caries at this point can be demonstrated it seems safer to allow this part of the cochlea to sequester rather than to remove it with the probability of opening the internal auditory meatus. Further removal of the upper portion of the cochlea also endangers the support of the facial nerve in the labyrinth. In cases in which this portion of the cochlea is perfectly loose it may be removed. My experience with operations upon the cochlea is limited to three cases, two of my own in which I successfully opened both whorls of the cochlea and removed the apex of the modiolus and one of Jansen's in which the cochlea was found to have sequestered and was completely and safely removed.

XVII.

CHONDROMA AND OSTEOMA IN THE FAUCIAL TONSILS.*

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It is my usual custom to make a pathologic examination of all the tonsils that I remove. In one pair I found something that played havoc with the microtome knife. On careful study of the sections I found considerable cartilage and bone growing within the structure of the tonsils.

A brief history of the case is as follows:

About the middle of last December, a nurse, 27 years of age, came in with her doctor and requested that I remove her tonsils. She gave a history of an attack of la grippe, associated with severe nausea, retching and vomiting, the month previous, but no history of trouble with the tonsils. In talking with her, I noticed a peculiarity of speech. She said that she had always talked so, but had paid no attention to it. On examination, the right tonsil was found larger than the left, although neither was especially large. In looking for the cause of her speech defect I could find nothing abnormal with the postnasal space or larynx, but the soft palate proved to be very firm and unyielding when she swallowed or pronounced certain letters. The only part of the soft palate that appeared to move was a small triangular area, about 4 mm. across, just above the uvula. The nurse and her doctor thought that there would be some relief of the difficulty of speech, if the tonsils were removed.

Under local anesthesia, swabbing with 10 per cent cocain, and hypodermic of 2 per cent cocain in 1 to 2000 suprarenalin, I performed a tonsillectomy. First freeing the right tonsil from the pillars and the supratonsillar membranes, with the

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*Presented before the Chicago Laryngological and Otological Society, April 14, 1908.

Pierce pillar knife, then applying the newly modified Boettcher-Farlow snare, the left tonsil was enucleated with the Pierce tonsil knife. Both tonsils were removed without any difficulty, but with the usual amount of hemorrhage. She stood the operation well, but before morning her former gastric troubles started up, with severe nausea and retching, which persisted for several days.

In gross appearance, these tonsils were not like the usual tonsil, large, compact and plump, but rather small with long thin processes, separated by broad deep crypts. The right tonsil, under the microscope, presents a broad band of cartilage and bone in the capsule, which extends into the broad fibrous septa that run down into the processes of lymphoid tissue, on each side of a deep crypt.

In studying the sections, of many different levels, the cartilage and bone were found to curve in, crowding through the lymphoid tissue, to meet each other, thus forming an irregular circle about 4 mm. in diameter, inclosing much lymphoid tissue surrounding the base of a crypt. There was always a dense fibrous band separating the cartilage or bone from the lymphoid tissue. There were other abnormal conditions about this tonsil. On the lateral surface, beyond the cartilage, there was considerable muscle structure. On the lower end of the tonsil there was a secretory gland, probably salivary. Several lobes of this gland crowded up, back of the lymphoid tissue, external to the lowest crypt, and crowded in between the cartilage and lymphoid follicles, that extended down into one of the processes. At the upper end of the tonsil a band of pure cartilage branched out from the cartilaginous circle and extended up along the capsule.

The left tonsil was sectioned in such a way as to make cross sections of several crypts. Back of these was a large triangle, 4 mm. across, made up of a broad fibrous band, the base of which forms the capsule, the point crowded up between masses of lymphoid tissue. Inclosed within this triangle were two smaller masses of lymphoid tissue, separated by a band of connective tissue. In this broad fibrous triangle were bands of cartilage, in which were seen many areas that had gone on to ossification.

On careful study of the cartilage and bone in these tonsils, I found the cartilaginous areas presented the typical structure of hyaline cartilage with lacunae, each inclosing a cartilaginous cell. Closely surrounding the cartilage was a band of fibrous connective tissue forming a perichondrium, and, with certain stains, notably Verhoeff's or Mallory's, one can see the surrounding fibers entering into and becoming a part of the cartilaginous structure. In many areas of the cartilage there were centers of ossification, which had developed into true bone, with their Howslip's lacunae lined with osteoblasts. With proper staining and careful study, one can see the apparent transformation of fibrous into cartilaginous tissue, passing on into bony structure. In some areas the bone is entirely surrounded by fibrous tissue, forming a periosteum, while in other areas the bone is in the midst of cartilage. The new elastic tissue staining method, just published by Verhoeff, demonstrates the formation of the cartilage and the development of ossification better than any other stain. Mallory's reticulum stain demonstrates the bone structure best.

A number of writers, notably Newcomb, Roe, Richards and Stirling, have reported difficulty in removing tonsils with the tonsillotome, finding it necessary to resort to bone cutting forceps to complete the removal of the tonsils. On examination, a single nodule of bone, supposedly from the styloid process, was found protruding into the tonsil. To reach the tonsillar region, the styloid process, which extends usually downward, forward and inward from the under surface of the temporal bone to an extent of two to three centimeters, would have to be elongated to more than five centimeters and curved forward and inward. Microscopically, this would present a single nodule of cartilage or bone and could be traced out through the structure of the tonsil. Palpation of the lateral wall of the pharynx would reveal the remaining stump of the styloid process, between the angle of the inferior maxilla and the vertebral column.

Another group of writers, namely, Carter, Kanthack, Lubarsch, Petroff, Pollak, Reitman and Toepfer, believe that the formation of cartilage and bone in the tonsils is due to metaplastic changes, of fibrous tissue into cartilage and then into bone. In the study of the tonsils in my case, the appearance suggests the possibility of just such a change, but to confirm this theory, there should be a definite clinical history and pathologic findings of chronic inflammation, such as these writers report, but which was lacking in my case. Senn, in his book on "Tumors" (1900) says: "The metaplastic theory concerning origin of bone is no longer tenable." "Tissue begets tissue of its own kind." There must be a matrix of embryonal cells as origin of development.

In the many cases reported by Coakley, Diechert, Halkin, Nosske, Orth, Roth, Ruckert, Walsham and Wingrave, the consensus of opinion is, that the cartilage and bone found in the tonsils, originated in abnormal development of cartilaginous rests of the second branchial arch. In the embryologic study of this second branchial arch, there is the dorsoventral cartilaginous bar, called "Reichert's Cartilage" made up of five segments. The stapes, by some, is supposed to be developed from the first segment. The second segment forms the base of the styloid process. The third segment forms the lower part of the styloid. It undergoes ossification later than the second segment and does not acquire osseous union with it, until adult life. Sometimes it remains separate throughout life. The fourth segment remains fibrous and forms the stylohyoid ligament. The fifth segment becomes the lesser cornu of the hyoid bone. The tonsil develops as masses of lymphoid tissue about an evagination of the lateral wall of the pharynx, in the third month of fetal life.

Cohnheim referred every tumor to its proper embryonic layer and claimed that a tumor never had its origin from mature tissue, but always developed from a matrix of embryonic tissue. He believed that during the process of cell differentiation in the embryo, groups of cells not utilized, or displaced, in the growth of the embryo, were arrested in their further development and remained latent until aroused to activity, when their proliferation resulted in the formation of a tumor. Hence, the matrix of embryonic cells is the essential cause of tumor formation, without which, all intrinsic and extrinsic causes are inadequate to produce a true tumor.

In these cases, then, where cartilage and bone have been found in the tonsils there must have been a displaced or unused matrix of embryonic cells. The second branchial arch furnishes us with the possible source of embryonic cells of this nature. In the same manner, we find cartilage in the parotid glands and about the external ear that originated from embryonic cells derived from the first branchial arch.

In the study of my case, I believe the following conclusions are justifiable:

1. The cartilage and bone are not part of the styloid process,

for we cannot palpate the stump of the styloid between the inferior maxilla and the vertebral column. Here, too, there is not a single nodule of cartilage or bone, but a ring or triangle.

2. The condition is not one of metaplastic changes, for there was no clinical history or pathologic findings that would cause such changes.

3. The logical conclusion, therefore, must be that the cartilage and bone found in these tonsils, was developed from a matrix of unused or displaced embryonic cells derived from the second branchial arch.

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BIBLIOGRAPHY.

Carter. Medical Record, 1905.
Coakley. Trans. Am. Laryn. Ass'n., 1904.
Diechert. Virchow's Archives, 1895.
Halkin. Bull. de Acad. royale de Med. de Belg., 1905.
Lubarsch. Virchow's Archives, 1904.
Newcomb. Trans. Am. Laryn. Ass'n., 1904.
Nosske. Cf. Centblt. f. Laryn., 1903.
Orth. Festchrift Virch., 50 Jahr, 1893.
Petroff. Beiträge zu Metaplasia Frage, 1901.
Richards. J. Am. Med. Ass'n., 1901.
Roe. Trans. Am. Laryn. Ass'n., 1904.
Ruckert. Virchow's Archives, 1904.
Stirling. J. Am. Med. Ass'n., 1896.
Toepfer. Arch. f. Laryn., 1902.

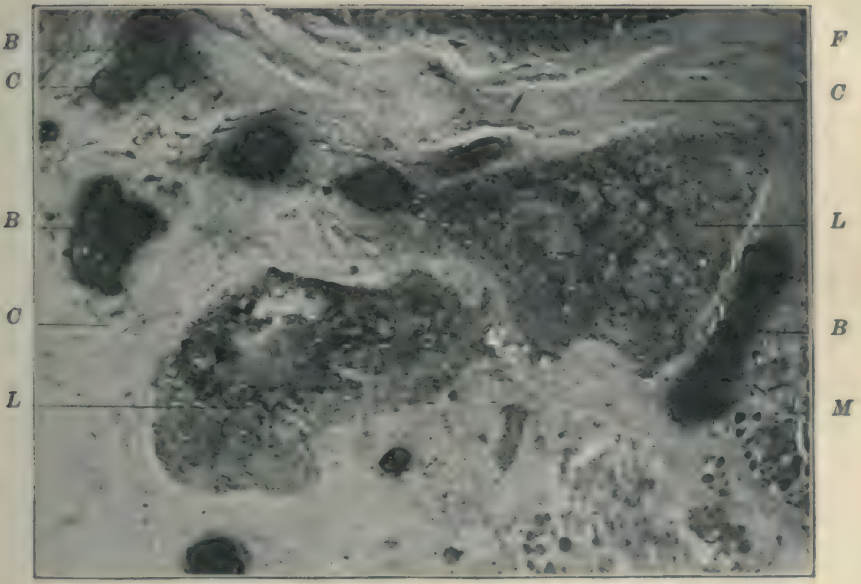

FIG. 1.—Fibrous triangle with areas of cartilage and bone. B—Bone; C—Cartilage; F—Fibrous connective tissue; L—Lymphoid tissue; M—Muscle. Hemotoxylin-eosin stain. Magnification 25.

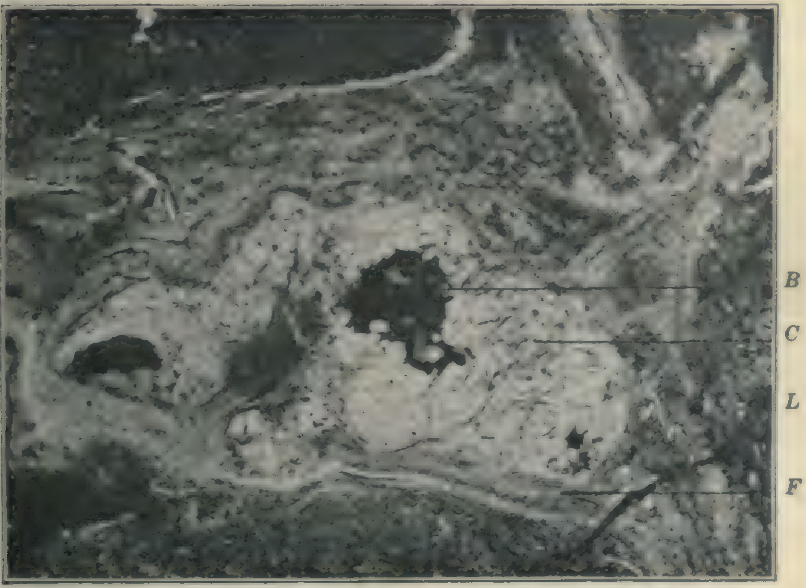

FIG. 2—Mass of cartilage and bone. B—Center of ossification. C—Cartilage. L—Lymphoid tissue. Verhoeff's Stain. Magnification 55.

XVIII.

DEVELOPMENTAL ABSENCE OF THE OUTER RIGHT SPHENOIDAL WALL OCCUPIED BY A VEIN COMMUNICATING DIRECTLY WITH THE CAVERNOUS SINUS. OPERATIVE FATALITY AND AUTOPSY.*

By Francis P. Emerson, M. D.,

Boston.

In the surgery of the accessory sinuses, brilliant accomplishment has been attended by unavoidable mishap. To overcome this or perfect the operation, various procedures have come to be known by their originators until today half a dozen definite and different plans are offered to accomplish the same end, some of them conservative and some the most thorough and radical that modern surgery can offer. The more that one studies anatomic specimens in connection with the sinuses, the more he is impressed with the large number of abnormalities and the necessity of exact measurements and familiarity with the various cavities of the head. Vital centers that are separated from pus cavities only by cribriform osseous plates lined by degenerate tissue are not ideal fields for operation. After several years of work in draining the sphenoid without anything suggesting an accident, the following condition was found and its attempted correction attended by a fatality:

In June, 1904, Miss L., 21 years of age, born in Massachusetts, and a school teacher by occupation, was referred to me by Dr. B. for treatment, with the following history:

Past History.—Severe attack of diphtheria at 5 years of age. Light attack of measles. Catarrh since childhood. The act of swallowing food was accompanied by gagging and vomiting which released a thick tenacious mass from the posterior nares. When actual vomiting did not occur, pharyngeal irritation excited hawking so that she has to leave the table. To avoid this she has been in the habit of using an anterior douche previous to each meal. There is pain over the bridge of the nose.

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* **Presented before the annual meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, January, 1908.**

Examination.—Anterior nares normal, except an enlargement of the left anterior turbinal cell of the middle ethmoid. Posteriorly there is a tenacious mass covering the entire pharyngeal vault. The posterior wall of the pharynx is glazed and dry. A small amount of dry secretion covers the anterior end of each middle turbinate. A bullet pointed probe shows a carious area in both sphenoids. The antra and frontal sinuses illuminate normally. General condition good.

July 5th.—The right anterior sphenoidal wall was removed with the Grünwald forceps, and the anterior end of the right middle turbinate with scissors and snare.

April 15th.—The left anterior sphenoidal wall was removed, together with the anterior end of the left middle turbinate. A carious area on the floor and inner wall was curetted.

March 24th, 1905.—No symptoms except dryness of the pharynx.

November 17th, 1906.—Came back complaining of a return of former symptoms, but less marked. A large cell was found containing pus, in the remains of the left middle turbinate which was opened and drained with biting forceps.

March 25th, 1907.—A thin tenacious grayish coating was found still present over the pharyngeal vault. Patient said that this still troubled her about twice a week. A thorough examination was again made and in the right sphenoid a carious area was felt on the outer wall with a soft mass posteriorly. On account of the previous operation this was taken to be polypoid, and but little force was used in probing, for fear of exciting hemorrhage. The middle turbinate and the anterior wall of the sphenoid had already been removed. A curette was introduced behind this presenting mass and gently withdrawn, but was followed by a profuse venous hemorrhage and a convulsion. The patient immediately became unconscious. A rapid tampon, after the patient had fallen in collapse on the floor, controlled the hemorrhage externally, but symptoms of brain pressure supervened, and she was removed to the Charlesgate Hospital, where she died at 11 o'clock, seven hours after the accident.

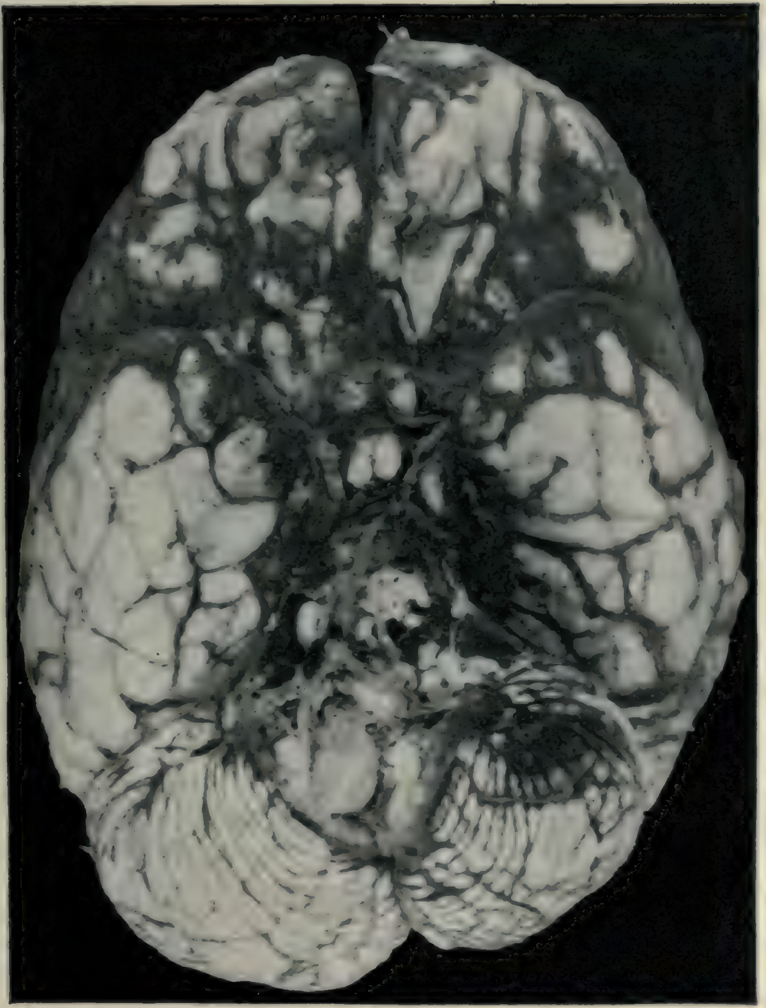
Autopsy by Dr. Leary, of Tufts Medical School, in the presence of Drs. Arnold, Courtney, Swan and others. A hemorrhagic clot filled the calvarium. A large varicose vein was found to occupy a dehiscence in the outer wall of the right sphenoid, which communicated directly with the cavernous sinus, and was the cause of death. The remaining ethmoid cells were everywhere necrotic, and about one-half ounce of mucopurulent secretion was found.

It was my custom in my early work on the sphenoids to break down the anterior wall (i. e., enlarge the normal opening with a Blake mastoid curette) until I found that this was apt to be followed by a rapid closure anteriorly before the cavity was dry. Latterly the Grünwald forceps that bite from behind forward, have proved more satisfactory and the drainage alone cures the case clinically. This is fortunate for if one studies a large number of sphenoid sinuses on the cadaver, he is at once impressed with the extreme thinness of the superior and outer walls which indeed may be cribriform, or so thin as to be readily seen through. The outer wall in addition may project into the general cavity so that it might be readily broken down in the radical Killian operation, unless one were working by careful measurements. It is easy to conceive that the dragging forward of a polypoid mass, even though there were no break in the osseous structure, might be accompanied by disastrous results.

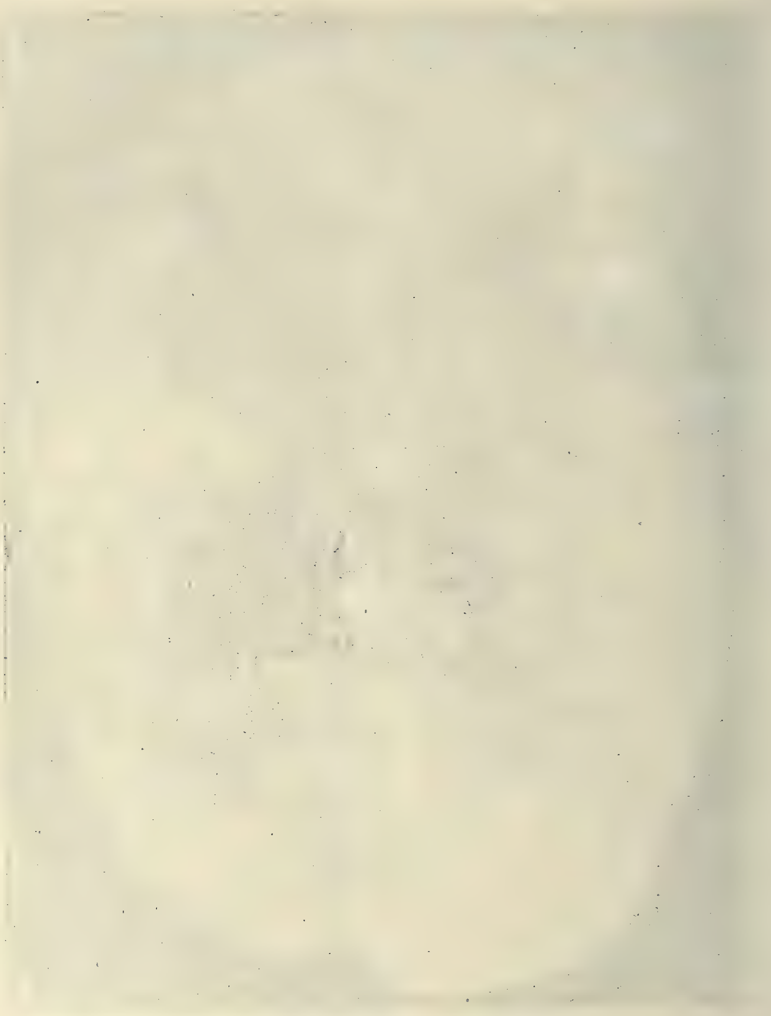
While clinical experience has taught us that the sinuses are rarely involved separately, there are a large number of cases that cease to give symptoms if properly drained intranasally. It is usual to associate disease of the sphenoid sinus with a glazed pharynx, a thick tenacious mass in the vault and dried secretion on the anterior surface of the middle turbinate. In women this is usually accompanied by an occipital or vertex headache. The diagnosis is readily confirmed by a small bullet pointed probe passed directly back to the pharynx through a line that passes midway between the anterior end of the middle turbinate and the septum. If it does not at once enter the osteum, slight elevation or depression usually at once locates it. The anterior end of the middle turbinate need not be disturbed unless diseased. It is unnecessary for diagnosis and generally is not required for treatment. The small probe with the shoulder which its bullet point gives it, will enter any osteum without special curve or measurement, as it can be manipulated very delicately, the bare bone being at once felt. Emphasis should be laid on the fact that removal of the anterior wall is all that is necessary to relieve symptoms. Cleansing is not only unnecessary, but is usually followed by a distressing headache. Other measures are dangerous and not needed. The removal of the anterior wall should be accomplished by the instrument producing the least trauma, and is therefore followed by the least reaction and tendency to fill in before the cavity is thoroughly drained.

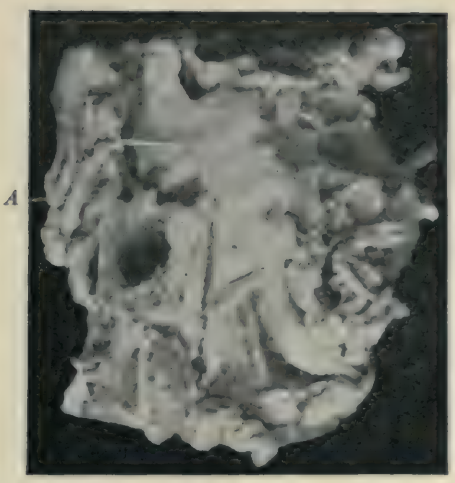
In the case cited, it was my intention to insert the curette behind the mass, entering from the inner side and draw forward. In doing so I did not feel any sensation of bony contact, which one is apt to recognize however slight the contact of a steel instrument with an osseous surface.

Dr. Leary's careful examination of the recent specimen showed an absence of the outer wall, and a large vein which communicated directly with the cavernous sinus, and which was mistaken for polypoid tissue. Later, after the specimen had been prepared by the prosectors, an undoubted fracture was found in the outer wall, and whether this was made at the time of the operation, or later in its final preparation, cannot be determined.



Subpial hemorrhage from vein in the outer wall of the sphenoid communicating with the cavernous sinus.





a. Developmental abscess of the outer wall of the sphenoid. Fatal hemorrhage from a large vein communicating with the cavernous sinus.

XIX.

REPORT OF A CASE IN WHICH DEFICIENT CEREBRATION WAS A PROMINENT SYMPTOM IN OTITIS MEDIA PURULENTA CHRONICA—CURED BY A RADICAL MASTOID OPERATION.

By D. W. Layman, M. D.,

Indianapolis.

A special form of diminished power of mental application, termed by Prof. Guye "aprosexia," is frequently noticed in children affected with postnasal growths, with or without exudate catarrh of the middle ear. We understand the cause of cerebral deficiency in these adenoid children, and know that under appropriate operative treatment such cases become normal, or are at least greatly improved, but I have never met with a condition such as aprosexia existing purely as the result of an otitis media purulenta chronica. It is familiar knowledge among otologists that such symptoms as "a marked change in the mental state, consisting of dullness and hebetude, seeming lack of thought or great slowness of thought, desire to be left alone and to keep the mind at rest, imperfect power of attention, defective memory of recent events," are all usual symptoms of intracranial lesions when they appear in cases of chronic purulent otitis media.

I wish to show, however, that the above symptoms can occur in a case of chronic purulent otitis media not intracranially complicated, but complicated with a chronic mastoiditis. I, therefore, present the history and treatment of the following case occurring in my hospital practice:

Harry H., aged 7 years, had scarlet fever when a baby, or a very young child. This was complicated with an otitis media purulenta. So far as could be learned, he had suffered from suppuration in the left ear since the attack of fever. When I first saw him the aural discharge was profuse and of very fetid and caseous character.

An aural examination in the fall of 1905 showed destruction of all but the upper part of the tympanic membrane; the handle of the malleus projecting into the perforation. On the posterior half of the tympanic cavity there was a polyp attached with a narrow base. Examination of the throat and nasopharynx revealed diseased adherent and submerged tonsils and small adenoids. Hearing was impaired, but not to a marked extent.

The ordinary symptoms of aprosexia, difficulty in sustaining attention, dulIness and slowness in school, were noticeable, but attributed at that time to the presence of the postnasal growth and submerged tonsils. Subsequent removal of these growths, however, proved that this was not the case. The growths were removed in the spring of 1906. The following year the child was quarrelsome, ugly and hard to manage in kindergarten; in fact, the teachers told me they dreaded the care of the boy on account of his ugly disposition. Besides, he was slow to learn, and evinced a seeming lack of thought and slowness of intellect, and showed a desire to be left alone.

His ear was treated with antiseptic irrigation and instillation of drops after the removal of the polyp. As this treatment proved to be of very little benefit, I decided to perform a radical mastoid operation. This was done in June, 1907. The operation was followed by an uneventful recovery.

A word or two about the operation.

The Schwartze-Stacke operation was performed, that is, the antrum, tympanum, attic and external auditory canal were converted into one cavity by removal of much necrosed bone, granulations, cholesteatoma and the remnants of the membrana tympani and ossicles, except the stapes. The natural meatus was enlarged by making an upper and lower flap of the cartilaginous meatus, by carrying a horizontal incision outward into the concha a distance sufficient to insure a good opening. The upper flap was sutured back over the dome of the excavation at superior rim of bone cavity. The lower flap turned down and sutured to the anterior lip of the curvilinear wound below. The mastoid wound was closed behind and healed by primary union. The wound was subsequently dressed through the enlarged meatus, and the entire surface dermatized nicely.

The operation was not performed with any idea of improving the child's disposition or mental state, but only with the view of ridding the patient of the suppuration in the temporal bone. The nurse at the hospital called my attention to the improvement in his mental state just prior to his leaving the hospital for the Orphans' Home. Afterwards I interested myself in this phase of the case, and asked the matron to notice whether she detected any change in the conduct of the boy. Three months after the operation she told me that Harry was like a different child, that he was bright, cheerful and took an interest in everything, and she said that the teachers reported that he was easy to manage and showed none of his old ugly disposition, and that he learned in school as readily as the average scholar. There was no improvement in hearing after the operation, but there apparently seemed to be, as he was very attentive.

We know that enduring forms of headache are caused by old chronic suppuration of the ear, especially with collection of exfoliated epithelium and granulation tissue in the cavities of the middle ear and spaces inside the temporal bone. Politzer mentions a feeling of pressure and heaviness in the head, or obstinate headache, as symptoms of chronic middle ear suppuration, especially in those cases complicated with mastoid disease. It is presumed that the symptoms mentioned by Politzer especially refer to cases of chronic purulent otitis media in adults. Children would less readily complain of a feeling of pressure and heaviness in the head, or obstinate headache: then, why could not these similar lesions in children produce symptoms which manifest themselves by all the signs of deficient power of mental application as in the case reported.

It might be asked, are these symptoms of deficient cerebration the result of a feeling of fullness in the head, or irritation due to more or less pressure on the brain by close proximity of the suppurating cavities, upsetting directly or indirectly the mental balance or nervous state, or, are they due to absorption of the toxines of pyogenic bacteria?

XX.

THE TREATMENT OF INTUMESCENT RHINITIS BY A SUBMUCOUS METHOD.

By Henry Horn, M. D.,

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(From the Universitatspoliklinik fur Ohren-, Hals-, und Nasenkranke, in Bonn a-R. Director: Geh. Med.—Rat. Prof. Dr. Walb.)

Intumescent rhinitis is of itself not a pathologic entity but the term clearly conveys to us the picture of a clinical condition which is of very common occurrence indeed. The etiology also is not clear, many throwing the causal burden entirely on the shoulders of a general bodily disturbance, such as alcoholism, circulatory, heart and kidney diseases, scrofulosis, anemia, etc., while others consider the condition purely a local one.

Probably the middle ground is nearer to the real truth of the case, and it goes without saying that where a physical examination revealed some general complaint, one would endeavor, as far as possible, to correct this condition in connection with the local treatment. On the other hand very few surgeons are in sympathy with that school which hold local measures, especially the employment of the galvanocaustic, unnecessary, and confine themselves to a general constitutional treatment only.

The brilliant results which have been achieved by the use of the galvanocaustic in the amelioration of this complaint are not to be denied. Where failures are found, one can usually explain them by a lack of a proper selection of the cases.

That every case of intumescent rhinitis can be benefited by the use of the galvanocaustic, and that every case of hypertrophic rhinitis cannot so be helped, places us in the position of asking what the dividing line is between these two conditions. Pathologically there is none, clinically an easy rule can be stated. If a turbinate with a smooth mucous membrane surface is easily reduced in size by cocain, it is favorable for the procedure. If it is slightly or not at all reduced, it is usually a case for a cutting operation.

The term smooth mucous membrane surface is used simply to exclude all polypoid degenerations of the membrane, all hypertrophies falling under the classification of raspberry (himmbeerartig), grape like (traubenartig), plicated (lappigenhypertrophy), which of course do not fall within the province of this paper, and are only to be treated by cutting or snaring operations.

In the splendid work of Ingals and Friedberg, on the "Treatment of Intumescent and Hypertrophic Rhinitis" in these ANNALS for March, 1906, we secure such a clear picture of the etiology, general course and clinical symptoms, that it is quite unnecessary to cover the same ground again. In a large clinic, where the general hygienic conditions of the patient are exceedingly unfavorable, where the mucous membrane tone is notably below par, where the material is enormous and constantly changing, where one is never certain of the patient returning for after-treatment; the disadvantages of the usual methods of cauterizing the inferior turbinate become quickly apparent.

The complications which depend far more upon the after-treatment and hygienic surroundings of the patient, than upon the operation itself, have been far more common with us than is shown by the statistics of the above mentioned authors, who perhaps included in their results many cases handled in private practice.

Not a month passes but one or more patients present themselves in the clinic with a septum-turbinate adhesion. The history almost invariably given is: that the nose was "burnt" because it was "stopped up," and that they had "never returned for after-treatment." The adhesions occur almost invariably in males and especially in persons with narrow noses and septal deviations. This is notably the case in all statistics, and seems to depend on the occupation, which by the inhalation of dust, chemical vapors, etc., brings about a local irritation, and rather speaks against a constitutional disturbance as the sole cause. Narrow noses with septal spurs, ridges or deviations are especially prone to lead to poor results by the old methods of cauterization.

Angina lacunaris, as first mentioned by B. Fränkel, is an exceedingly common complication, coming on about the third day and often occurring in spite of the most careful after-treatment.

Septum-turbinate adhesions are among the most disagreeable of all the complications which have their origin in this procedure, and is by no means as infrequent as the statistics of the above mentioned authors would indicate. In narrow noses combined with sharp crests, an accidental burning is sometimes unavoidable and the subsequent results far from satisfactory. Just here another point arises which has only made itself felt since the introduction of the submucous resection of the septum; no septum operation is pleasant where the mucous membrane is scarred through a previous operation or an accidental burning of its mucous membrane, and no septum operation fulfills its function if we leave the swollen turbinates untreated. The preliminary cauterization of the turbinates seems the rational procedure.

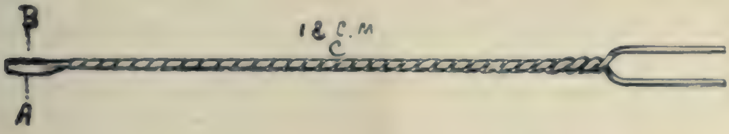
The submucous caustic of the inferior turbinate, when properly carried out offers an ideal solution out of all of these difficulties.

Some years ago a procedure, generally ascribed to Grünwald, was proposed along these lines. This consisted in entering the anterior end of the turbinate with a glowing point and gradually undermining the mucous membrane as far back as possible. The practical result was that at the point of entrance a large burn was made, and at the posterior end a second perforation resulted. If the membrane was at all adherent at any point, a third perforation was made, until finally during the healing process, the intervening mucous membrane would slough away and an open wound result.

The next step was reported from the Hajek Klinik in a short note of ten lines. Here the anterior end of the turbinate was entered with a knife, the membrane undermined, and an ordinary pointed burner introduced cold and withdrawn hot. This procedure also had its disadvantages. The sharp knife often perforated the membrane in the middle third and at best only cut through the intumescent vessels, leaving an unsatisfactory passage for the point to follow. If the swelling was not marked or if, as it often happens, the middle third was slightly adherent, the unprotected membrane would be burned through and again we would have to deal with an open wound.

The submucous method, about to be described, does away with all these disagreeable features and has as well the following great advantages:

1. Complete healing takes place in from three to five days.
2. The swelling after the operation is practically nil.
3. The mucous membrane remains intact throughout its entire length.
4. The wound is aseptic. The entrance point closes of itself.
5. Adhesions with the septum are impossible.
6. The physiologic functions of the mucous membrane are preserved.
7. The burning takes place only in that tissue which it is intended to destroy: The cavernous tissues of the turbinate.
8. The shrinkage is complete along the entire length of the turbinate, and an ideal and permanent air space is the result.
9. So far no complicating tonsillitis, middle ear inflammations, or adhesions have been observed.
10. Burns of the septal membrane are absolutely avoided.
11. The disagreeable smell of the burning tissues and the occluding smoke are done away with.
12. If the patient fails to return for treatment no harm can result.



Operation.—A cotton tampon moistened with a 20 per cent solution of cocain is laid in the nose for a few minutes until the turbinate is entirely free from feeling as tested by the knife point. If the intumescence is not great and the cocain promises too great a shrinking, a similar solution of alypin can be used.

A small perpendicular incision is made through the membrane at the anterior end of the turbinate. With a Freer septum elevator, the mucous membrane is dissected free from the bone as broadly and as far back as possible. The use of a large elevator or a knife is not to be recommended, as a perforation is easily made.

The form of the burner is of great importance. As shown in the illustration, I use the so-called aseptic burner (C), 12 cm. long, with a protected burning wire. The part (a) is of very heavy platinoiridium wire 0.7 mm. in diameter and 1 cm. long. The part (b) remains cold during use and protects the overlying mucous membrane. The rheostat is set beforehand at almost a white heat, as the blood under the upraised membrane causes a rapid cooling of the point, thereby allowing it to become fastened to the membrane and perhaps causing a tear.

The burner with the platinum turned toward the bone is introduced cold to the posterior end of the pocket formed by the upraised membrane, withdrawn a few millimeters, to prevent perforation of the end pocket, and the current turned on. The burner, always closely and firmly pressing the bone, is slowly withdrawn. Just before the entrance is reached the current is shut off, and the burner quickly removed.

The overlying mucous membrane, being protected by the form of the burner, is not injured, but immediately and firmly contracts and subsequently becomes firmly attached to the bone. The vessels are permanently destroyed thus greatly protecting against a recurrence. Over a year has elapsed since some of my first cases were operated by this method and a present examination shows the same condition as was seen two weeks after the operation.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINOLOGIC AND LARYNGOLOGIC LITERATURE.

I.—EAR.

Report of Cases of Brain Abscess.

F. E. Kittredge, Nashua, N. H. Case one was a full-blooded Indian, 9 years of age, in which the brain abscess followed about a month after an operation for a purulent mastoid and an epidural abscess, recovery from which had occurred. This was followed by severe headaches, violent attacks of vomiting, slightly subnormal temperature and a pulse of about 60. The original incision being reopened, the dura was found to be in a healthy condition but somewhat bulging. Laying open the membrane, went in about one inch and a half with a needle and pus was found one and one-half inches deep in two pockets. Both cavities were drained with gauze; about one and a half ounces of pus being evacuated. The case did well for three weeks when meningeal symptoms developed, followed by unconsciousness and death. After the development of the meningeal symptoms, a thorough examination was made with the finger which seemed to break into the wall of the lateral ventricle and upon the withdrawal of the finger, water and pus of considerable quantity, both came away. The author estimates that one-third of the hemisphere had disappeared.

Case 2.—Male, 30 years of age. Simple mastoid first done. Afterwards had a severe convulsion followed by drowziness, vomiting, temperature of 99, pulse 60. Brain abscess diagnosed, trephined about one inch above the zygomatic ridge; bulging dura, pocket of pus one-half inch from the surface. Bone opening enlarged and good drainage secured with gauze. Recovery about three weeks after operation.

Case 3.—Male, 27 years of age, slight discharge but no mastoid symptoms. Temperature 102 to 105, delirium, unconsciousness, meningitis; brain abscess suspected. Trephined through the right temporo-sphenoidal region just above the ear, dura opened, brain tissue incised, two ounces of pus evacuated. Death in three days. No relief having occurred except of the headache.

Case 4.—Male, 16 years of age. Severe headache, temperature 101, pulse 55; mastoid symptoms. Operated for the mastoid. Headache no better, slow pulse, vomiting. Brain abscess diagnosed. Dura opened after trephining; no pus found in the zygomatic region. Trephined over the cerebellar region and explored with trocar, nothing found. Symptoms not improved at all and after several days with a temperature ranging from 98 to 100 and pulse 45 to 55, further search for pus was made, by opening the original mastoid cavity between the semicircular canal and the anterior part of the cerebellum; no pus was found. The openings were packed with gauze and there was considerable discharge of serum. Headache improved, pulse went to 60. A day or two later there was marked nystagmus in both eyes followed by temporary paralysis in the left side of the face. Recovery took place. Whether this last particularly belongs with the brain abscess cases may be a question. *Richards.*

Pathologic Anatomy of Labyrinth Inflammation Extending From the Middle Ear.

W. LANGE (*Beiträge z. Anat., Phys., Path. u. Therapie d. Ohres. der Nase und des Kehlkopfes,* Bd. 1. Nos. 1-2) reports in considerable detail the histologic findings in 14 cases of labyrinth suppuration. He found that in 9 of these cases the point of primary infection was through the semicircular canals; in 4 through the oval and round window; and in 1 through both the semicircular canal and the windows. In acute cases the windows seemed more frequently to be the primary points of entrance than the semicircular canal. Polyps and excessive granulations are most frequently the predisposing conditions. Cholesteatoma by causing erosion of the semicircular canal open a gateway through which an acute infection may at any time find entrance to the labyrinth. As to extension of the disease within the labyrinth, Lange found that in all his cases where the membranous labyrinth was necrotic, the ductus endolymphaticus showed more or less involvement. The soft parts of the ductus were generally necrotic almost to the external aperture of the aquaeductus vestibuli. In 4 cases the propagation of the inflammation through the cochlea was stopped by old inflammatory deposits in the scala tympani, and in one case recent changes had completely obstructed the lumen of the scala tympani. In 8 of the 14 cases pus was found in the internal auditory meatus The function of the facial nerve was destroyed in only one case. There are a good many references mentioned in this article but they are all from German literature. *Wood.*

Closure of Bone Defects After Antrum Operations.

A. PASSOW (*Beiträge z. Anat., Phys., Path. u. Therapie d. Ohres. der Nase und des Kehlkopfes,* Bd. 1. Nos. 1-2). For the closure of old bone fistula following mastoid operations, Passow suggests the following technique: The scar is split throughout its length by an incision behind the ear, following the first operation. This incision should be carried completely through the depressed cicatrix, and the skin elevated from the wound. The bone wound is then carefully freshened with a curette. The skin around the wound is undermined for a considerable distance and carefully separated from the connective tissue and periosteum. The periosteal flaps are now carefully raised, one in front, one behind, the wound. The flaps whose bases are below must correspond in size to the breadth and depth of the wound. These flaps are now sutured in the middle line with catgut, and pushed into the cavity in the bone. The layer of connective tissue and periosteum posterior to the wound is thin, so that in this direction the broadest possible flap should be obtained. The flap from near the insertion of the ear is very narrow but generally rather thick. The scar tissue of the skin-wound is removed with scissors and the skin edges then brought together, preferably with Michel's hooks. This method of utilizing the periosteum, according to Passow, rarely fails to close in these post-operative defects in the bone. *Wood.*

Anatomy of Congenital Deafness.

FERDINAND ALT (*Monatsschrift für Ohrenheilkunde sowie für Kehlkopf. Nasen. Rachenkrankeiten,* February, 1908) reports a case of congenital deafness, in which he made a careful and complete histologic examination of the ears. The patient had died from tuberculosis. The case was one of complete deafness on both sides. The middle ear was practically normal, and the labyrinth followed out in general the ordinary scheme. The terminal elements of the nerves were only par-

tially developed, and further had subsequently undergone a marked atrophy. This condition was seen in the vestibules in all of the sensory epithelium of the cochlea, and also there was a collapse of the membranous labyrinth. The nerve itself in the region of the internal auditory canal showed less atrophy than in the individual nerve canals and the sensory epithelium. There was no sign of any inflammation either recent or old.
Wood.

Retroauricular Opening After Radical Mastoid Operation.

Ernst Winckler (*Arch. f. Ohrenheilk.*, Bd. 75, Nos. 1 and 2) makes a plea for the establishment of a permanent opening back of the ear, following the radical operation. He says it is especially indicated if there is any labyrinth complication, fistula of the semicircular canals, or disease of the promontory, making a careful observation of these structures necessary after the operation. Further, it is very useful when the cells surrounding the Eustachian tube are involved, and also in cases of complete exenteration of the middle ear which before they have healed come into the hands of another physician. This retroauricular opening affords the second physician a good chance to look over the operative wound. In tuberculosis of the middle ear the retroauricular opening is absolutely indicated, and also where there is cholesteatoma present.

Winckler gives in detail his method of operating for the establishment of a permanent opening. *Wood.*

Acute Labyrinthitis.

G. Alexander (*Archiv. für Ohrenheilkunde,* Bd. 75, Nos. 1 and 2) reports four cases of inflammation of the labyrinth following the radical mastoid operation. The chief symptoms were nystagmus toward the diseased side, dizziness, and a loss of equilibrium coming on within 24 hours after the operation. Also, in two cases there was some meningitis. The dizziness and loss of equilibrium cleared up in a few day, but the nystagmus generally persisted for a week or more. The rapidity of the cessation of the symptoms differentiated these cases from purulent labyrinthitis, and it is most probable that the condition present was a circumscribed serous labyrinthitis. While in none of the cases was the labyrinth visibly injured before or during the operation, it is most probable that the unavoidable trauma of the middle ear was the important etiologic factor. *Wood.*

Bier's Hyperemia Treatment in Acute Middle Ear Suppuration.

F. Isemer (*Arch. für Ohrenheilkunde,* Bd. 75, Nos. 1 and 2) reports in detail nine more cases of otitis media treated by Bier's passive hyperemia. From a study of these cases he arrives at slightly different conclusions than formerly. He says that treatment of otitis media with passive hyperemia is not void of danger, as in employing this therapy the symptoms became so markedly lessened that it is not an easy matter to know when to institute surgical interference. Hence he does not advise its use by the general practitioner. Streptococcic and diplococcic infections are especially dangerous. The hyperemia works better in staphylococcic infections and especially in cases with mastoiditis where there exists a cortical fistula. Here an incision through the soft parts gives a chance for the pus to escape. Relief from pain is the most striking result of the hyperemia treatment, but this in some cases may be exceedingly dangerous. This treatment should never be used in cases with intracranial complications.

Wood.

II.—NOSE.

Pansinusitis Nasalis with Fatal Course.

H. J. L. Struycken (*Tydschr. v. Geneesk.,* May 11, 1907) reports the following: Consulted in October, 1900, by a 48-year-old man for right-sided otorrhea, headache, nasal obstruction and mucopurulent discharge from nose and postnasal cavity. In right and left nose many polypi, between which pus constantly dripped. Left frontal sinus less transparent than right one. Inflamed remains of tonsils and adenoids. Paracentesis of the drum was done. Pain disappeared and temperature became normal. A foreign colleague had to remove the polypi; a serious secondary hemorrhage with a complicating bronchitis kept patient in bed for weeks. He returned in 1901; the aural inflammation on the right side was cured with cicatrices adherent to the promontorium, but purulent secretion from the left middle ear was now present and purulent secretion of the left conjunctival sac. The nasal polypi had returned; asthmatic oppressiveness and dizziness present. After removal of the remains of the tonsils and the polypi, the pus appeared chiefly to be secreted from the left side. This frontal sinus could be easily probed; the knob of his (SH[5]) magnetic sound could be demonstrated 1.5 cm. above the brow. The canula was easily introduced and with the first lavage fetid pus was discharged. The general condition improved markedly, so that against his insistant advice the patient refused any further operation; the secretion from the left ear had stopped, but not from the ethmoid and frontal sinus. He then returned once a year for removal of the obstructing polypi. He went through a serious pneumonia, which weakened his heart; the attacks of headache increased and his general condition grew worse, he became apathetic; rheumatic pains in back and limbs. A year and a half later Struycken was called for serious facial pain and protrusion of the left bulb. The eye was projecting about 1.5 cm., slight chemosis, painful swelling in the inner upper corner of the orbit. Irisreaction and fundus normal; all ocular motions possible with restriction. Polypi and pus in the nose. Violent pain in the forehead at the left side and around the eye for about six days; temperature was 36.4°. Next morning the left frontal sinus was opened; it was filled with fetid pus and also some accessory cavities behind the brow. The entire anterior and lower walls were removed and a large opening made in the nose by resecting the ethmoid cells. The origin of the protrusion was not found. The following day the temperature was 38°; less pain in the left forehead, but pain in the left upper molars and also the right temporal region. The maxillary sinuses showed less transparency than formerly; after trephining the left one and perforating the septum, which divided this cavity in an anterior and posterior part, cheesy pus was washed from the posterior cavity. This cavity was connected with the ethmoid cells. The second day, temperature was 37.5°; slight bronchitis; much purulent secretion from the ethmoid cells; protrusion slightly diminished; slight general stiffness of the muscular apparatus; euphoria. The third day the temperature was 39°. The entire ethmoid was removed with the medial wall of the orbit; the superior wall of the orbit as far as the fissura orbitalis superior consisted of a series of non-communicating bone cells filled with thick yellow odorless pus. The sphenoid, which showed no changes, was not opened; the antrum was free from pus. Temperature decreased during the evening to 37.3°, patient felt well without any pain. Next day temperature was again 39°. The protrusion was much diminished, the eye less movable, but nearly insensible to touch; iris-reaction normal; the arteries in the fundus less filled, veins somewhat wider. No bacteria in the blood, many multinuclear leucocytes. No swelling of the spleen. Patient became delirious. The following day the confusion increased, tongue was dry. Respiration slightly of Cheyne-Stokes type. Stiffness of the neck increased. At the right side now and then spontaneous contractions; the general muscular force at that side has decreased. This right side of the body was paralyzed on the following day; coma increased; left pupil wide without reaction. The 8th day after Struycken called the temperature was 39.2°. The left frontal lobe was explored, the dura looked normal, no pus after puncture. On the right side, frontal sinus and ethmoid cells were opened and found filled with pus, without direct communication with the brain cavity. In the afternoon the temperature arose to 41.2° and respiratory paralysis resulted in death. Section showed the convexity of both hemispheres opaque; at the base of the pons some flakes of pus. On section the ramus ophthalmicus nervi trigemini bathed in pus and the vena ophthalmica was closed by a non-purulent thrombus. The nerve, entirely softened by pus, could be followed as far as the brachium cerebri ad pontem, where a focus of the size of a hazelnut was found, the pons itself being narrow. No other abscess formation in the brain. Some small bone cavities were found in the neighborhood of the glandula pinealis and of the fissura orbitalis superior, filled with pus; these last ones, which were not opened during the previous operations, seemed to be in direct communication with the ramus ophthalmicus. Both the sphenoid sinuses were free. The other nerves, as far as visible in the cerebral cavity, were not infiltrated with pus. Probably it would have been better to have opened at the right and left side all bone cavities in one sitting, but patient was afebrile and it was possible that edema of the retrobulbar tissue caused the protrusion. The euphoria and decrease in temperature after the second operation set at ease and did not encourage further manipulation as the opening of the different cavities in the roof of the orbit took much time and exertion. To expose the dura mater is not at all an indifferent question, when these cavities are infected, as meningitis easily follows. But even the most radical operation would not have succeeded here. *Blaauw.*

Purulent Bone Cyst of the Inferior Turbinate.

JACOB COHN (*Monatsch. f. Ohren.,* February, 1908) reports a case of bone cyst of the inferior turbinal which was probably congenital. It had lately become inflamed, and a fistula formed on the upper surface of the turbinal through which it had discharged. The cyst had no connection with the maxillary or antrum. *Wood.*

Submucous Resection of the Nasal Septum.

GUSTAV KILLIAN (*Beiträge z. Anat., Phys., Path. u. Therapie d. Ohres., der Nase und des Halses,* March 10, 1908) suggests for anesthesia in submucous resection of the nasal septum, a perineural injection of cocain, which he says can be carried out in almost every instance. The nerve which supplies the greater part of the septum is the nasopalatine nerve, from Meckel's ganglion. The front part of the septum is supplied by a branch of the nasal nerve; hence four injections are required, one at the base of each of these nerves or each side of the septum. Killian says that the injection is very easily made except on the convex side in a bad deviation. For making his solution he uses a tablet of adrenalin and cocain. When it is not possible to inject the nasopalatine nerve at its origin on the septum the nerve may be reached further down its course. Ten minutes should elapse after the injection before operation is begun.

In relation to operating on young children, Killian says that with instruments smaller than those used for the adult the operation is fairly easily carried out under general narcosis. He has operated on children as young as four years of age.

Wood.

III.—LARYNX.

The Significance of Tuberculosis Laryngis for the Treatment of Tuberculosis Pulmonum.

PROF. H. BURGER (*Tydschr. v. Gen.,* Feb. 2, 1907) mentions the laryngeal tuberculosis as the most frequent complication and the most important clinically of the lung tuberculosis. He thinks that it occurs in about 30 per cent of the cases. There is no doubt regarding cure of laryngeal tuberculosis, but there is much difference of opinion as to the number. Nearly always the primary lung affection is of the highest importance for the prognosis; in many cases the amelioration or impairing of the larynx goes on equally with the lungs. A permanent cure can only occur when the tuberculous tissue in both organs is changed into cicatricial tissue. Cure of a larynx tuberculosis without local treatment, only dietetic-climatic, is only very rarely seen. Burger only saw temporary amelioration, but never a spontaneous local cure; but he saw more than once patients return from a climatic or sanatorium cure with very much better lungs, while at the same time a slight laryngeal affection had become worse. A tuberculous larynx has to be treated after the rules of the modern tuberculosis therapy with functional rest, that means to remain silent, and as not less important, the local or surgical treatment must be instituted. This includes application of chemical agents to the tuberculous ulcera, the most important of which is lactic acid, the scraping of ulcerated surfaces, the removal of granulations, the cutting away and burning galvanocaustically of granulations and infiltrates.

Three histories follow, which prove that consequent treatment will give satisfactory results:

1. A clergyman, 44 years old, consulted Burger September 10, 1898, for slight complaints in swallowing and now and then hoarseness, no cough. Some years previous he had a serious lung disease with tubercle bacilli in the sputum. His thorax was flat, respiratory sounds in both apices weak, but no rhonchi. Laryngoscopically an irregular ulcerative swelling at the posterior wall of the larynx.

Treatment.—Four times, application of 10 per cent carbolglycerin (October 30-November 3, 1898), cutting away of the swelling with the double curette (November 4) and 30 times, application of 30 per cent carbolsulforicinol (November 20-December 20, 1898). This treatment did not produce any reaction. At the end the posterior wall had entirely cicatrized. The cut away pieces contained tuberculous tissue with cheesy tubercles. On July 30, 1900, a small raised point is visible, which is cauterized for security sake. July 17, 1901, acute laryngitis with red vocal cords and light swelling of the posterior wall.

2. A 22-year-old nurse began treatment in December, 1895. She began suffering four years previous with cough; spat blood twice, one and five years ago, sometimes pain in throat, increasing hoarseness. She was weak, had night sweats, was emaciated and had pains in back and thorax. Aphonia through ulceration of the left vocal cord with granulations, also the anterior half of the right vocal cord, while the under side of both cords was much swollen. The posterior wall was free. Infiltration of apex of the left lung and tubercle bacilli in the sputum. The treatment, while in the hospital, was difficult through lack of appetite, tendency to vomiting, hypersensitiveness of the larynx, insufficient action of cocain and the additional hysterical temperament of the patient. Besides general measures, the larynx was frequently treated galvanocaustically and with pure lactic acid; the treatment lasted for about a year. The larynx looked then normal except a slight redness and swelling at the posterior part of the left vocal cord. During 1897-1902 a few circumscribed recurrences appeared, always at the posterior part of the left vocal cord, which healed with pure lactic acid. The last treatment was given in May, 1902; two galvanocaustic applications. Since the larynx has remained normal, the apex of the lung has cicatrized. In 1904 and 1906 she went through a full term pregnancy, both times the danger spot at the posterior end of the left vocal cord showed a slight redness with very slight swelling without any other disturbance.

3. A 22-year-old, hired girl, from a tuberculous family, came in April, 1895, under treatment for tubercular infiltration of the apex of the right lung, a scrophuloderma of the left cheek and a high degree of laryngeal tuberculosis; ulcerating infiltration with granulations of the entire right half, the vocal cord could not be discerned, the posterior wall was moderately thickened. The treatment consisted in removal of the new formation and applications of pure lactic acid, which was often disturbed by new formation of the removed granulations. The last curettement was done November, 1895, and the first of December the patient was discharged. The applications with lactic acid were continued till February, 1896. Since then no treatment. The larynx looks normal, so that it is even impossible to recognize the previously diseased side; the lung also remained cicatrized.

These cures of 8, 10 (4) and 11 years' standing illustrate the possibility often doubted of lasting success in laryngeal tuberculosis. The last two cases are examples of very important tuberculous affections, with very dubious prognosis. After discharge from the hospital both patients took up again their hard work. The voice regained in all three cases its normal sound. To make the focus in the larynx harmless will have a supporting influence for the cure of the lung condition.

The surgical treatment demands not only a complete technic, but also a large amount of patience, perseverance, faith. Who retires to the camp of the nihilists, disheartened by a dozen disappointments and failures, cannot judge fairly regarding the efficiency of this treatment. Admission to this sanctuary only is allowed the faithful. Another cause of failure is the difficulty and still the necessity of a long lasting treatment and control during months and years.

High altitude is not unfavorable for laryngeal tuberculosis. But a patient with laryngeal and lung tuberculosis should not be taken in any sanatorium, unless technically well trained laryngological help can be given. The family physician never should send a tuberculous patient away without an examination by a competent laryngologist for detection of a latent laryngeal tuberculosis. *Blaauw.*

IV.—PHARYNX.

Something About Hemorrhage After Tonsillotomy and a New Method of Relief.

I. C. HENKES (*Tydschr. v. Geneesk.* January 26, 1907) describes first the different anatomic possibilites, which may lead to a hemorrhage and all the usual ways of checking. He then describes his method, which is based on principle of Heerman and Escat, who bring the posterior arc in contact as much as possible with the anterior by means of a strong silk suture. Henkes had made clips, similar to those used in surgery, but larger and of thicker metal. The distance of both sharp points is about two centimeters. Forceps were made for depositing the clips at their place; its tube can easily be turned 180 degrees, so that it can be used for right and left side. Another forceps was constructed for removal of the clips, so bent that the hand does not obstruct the view; its ends are bent over a length of about 1 cm., flattened in the sagittal direction, so that this part, when the forceps are closed, can be easily pushed between the outer side of the palatal arches and the inner side of the closed clip. Henkes used this method in a case of a boy, 16 years old, from whom he removed both fibrous tonsils with the tonsillotome. The hemorrhage stopped on the left side spontaneously after a few minutes and on the other side after finger pressure during half an hour. In opposition to advice he took solid food the next day, which produced a pretty strong parenchymatous hemorrhage between the palatal arches. Every method failed to check it, until two clips were introduced 1 cm. lower than the first, a silk thread around each was glued with adhesive plaster to the cheek. The manipulation lasted about five minutes. The slight pain disappeared slowly. An iodoform gauze tampon was before put over the wound; this was removed with the clips after 24 hours. No recurrence. *Blaauw.*

Treatment of Peritonsillar Abscess.

MOLL (*Tydschr. v. Geneesk.,* October 13, 1906). The treatment is incision if an abscess is present, or otherwise a Priessnitz-bandage and wait till an abscess has formed. Moritz Schmidt advised incision even if no clearly defined abscess is present. This is not advisable for the general physician. The pus shows itself first in the fossa supratonsillaris behind the anterior pillar. When the patient puts his tongue out and sounds "e" the space between the pillars may be seen when the cheek is stretched. A strongly bent sound may penetrate behind the anterior pillar after cocainization and nearly always pus is found.

Blaauw.

ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY.

VOL. XVII. SEPTEMBER, 1908. No. 3.

XXI.

RECURRENT AND ABDUCTOR PARALYSIS OF THE LARYNX. INTRODUCTORY REMARKS ON ANATOMY AND PHYSIOLOGY. ETIOLOGY OF PARALYSES OF CENTRAL ORIGIN.*

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The subject, selected by your Council for general discussion, is in my opinion the most interesting and at the same time the most difficult one in the whole domain of laryngology. It is interesting, because it leads the laryngologist at once beyond the sphere of his specialty, and it is difficult, as it deals with the most intricate questions of neurology, about which in many instances the investigators entertain conflicting opinions.

For a full understanding of the part assigned to me—the central causes of recurrent and abductor paralyses—a clear conception of the motor centers in the cortex and bulbus, of their predominant functions, their connecting strata, is absolutely necessary. With the approval of your presiding officer I shall briefly analyze the anatomic features and add such physiologic and pathologic data, as have a direct bearing on our subject.

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*Read before the American Laryngological Association at the 30th Annual Congress, Montreal, May, 1908.

I.—REMARKS ON ANATOMY.

The larynx has to perform two functions, diametrically opposite each other, viz., phonation and respiration. The chief center for phonation, adduction of the cords, a voluntary act, is in the cortex; for respiration, abduction of the cords, a vital process, in the bulbus. In addition there are subordinate centers in reverse order and of lesser dignity for phonation in the bulbus, for respiration in the cortex. The connecting nerve fibers run from the cortical to the bulbar center and vice versa, through the internal capsule. The appended diagram from Rethi[1] shows very plainly the different motor centers and their relationship to each other.

The cortical motor center for phonation was discovered by H. Krause[2] to be located at the descending surface of the prefrontal convolution. The result of his investigations was confirmed by Semon and Horsley,[3] [4] who by extending their researches found additional motor areas in the cortex for acceleration and for deepening of the respiration. By ingenious experiments, Risien Russell[5] succeeded in finding below the phonatory center a locality from which abduction of the cords can be elicited. The illustration of the different cortical motor centers is a copy from Semon's treatise in Heymann's Handbook of Laryngology and needs no further comment.

The bulbar representation of the larynx is in the fourth ventricle, and we find in the alea cinerea two localities, irritation of which produces typical abduction of the cords, the respiratory act; further, smaller areas for bilateral abduction, one for unilateral abduction and one for the cadaveric position of the cords, all of which are graphically shown in the illustration, which is also taken from the same treatise by Semon.

The existence of the subordinate centers, mentioned above, has been proven by repeated experiments on animals, of which one only will be cited. Acephalic monsters and animals, whose brains have been extirpated, are able to utter a cry, due to the subordinate phonatory center in the bulbus, but their respiratory movements are not materially influenced, confirming the bulbus to be the main center for respiration.

These elementary anatomic data, condensed within the narrowest possible compass, have almost universally been accepted; the first real discrepancy of opinions we meet, is about the question of the root of the motor nerve of the larynx, which is not definitely decided at the present day.

After the careful experiments of Schech[6] the laryngeal motor nerve was for many years considered to have its source from the inner branch of the accessory Willisii, till Onodi[7] and Grabower[8 9] brought forth forcible arguments in favor of the pneumogastric, exceptions to which were taken by some authors. But a recognized authority, H. Oppenheim, says (1905) in his book[10]: there is at present no certainty in regard to the participation of the accessory nerve in the innervation of muscles supplied by the pneumogastric.

The illustration from Edinger shows very clearly the intimate relationship of the nerve nuclei, the motor fibers being in black, the sensory fibers in red color.

Another much debated issue relates to the nature of the recurrent nerve. Whilst many investigators strongly believe it to be exclusively a motor nerve, others offer good reasons, based on animal experiments, to consider it a mixed nerve, carrying also centripetal fibers. But if the latter view should prove to be correct, we cannot yet draw any definite conclusions from it in regard to the human system, as even in animals a differentiation exists in the response in different species.

The last and a most important anatomic fact is the discovery by Risien Russell[11] of the existence of special nerve fibers for adduction and abduction, which run in separate bundles throughout the whole course of the recurrent, those for the abductors on the inner, those for the adductors on the outer side of the nerve trunk. They can be dissected to such an extent, that one set of fibers responds to electrical irritation exclusively by adduction, the other exclusively by abduction of the vocal cords. By his work the formerly vexed question was solved, how a supposedly homogeneous nerve could effect such directly antagonistic movements.

II.—REMARKS ON PHYSIOLOGY AND PATHOLOGY.

The cortical motor centers of the larynx differ from other cortical motor centers in the essential point that irritation of either one is always followed by symmetrical bilateral adduction of the cords, phonetic movements, which also take place when one cortical center has been destroyed by disease or experimentally removed. This physiologic observation had enjoyed universal acceptance, till Masini[12] maintained that he found a locality in the cortex, from which movements of the opposite side of the larynx could be produced. Subsequent experiments by others did not verify his statements, but recently Katzenbach[13] obtained by an improved method similar movements of the corresponding as well as opposite side.

The influence of the bulbar center on respiration and on the position of the glottis has been carefully studied by Semon[14] in his endeavor to ascertain if the vocal cords play an active part during quiet respiration, and to determine the cause of the difference between the width of the glottis during life and its cadaveric position after death or after severance of the pneumogastric or recurrent. He found by numerous and carefully made measurements with graduated mirrors, that in over 80 per cent of adults the glottis remains stationary during quiet respiration and is considerably wider than in the cadaveric position. Its average width is in men during life 13.5 mm.; after death 5 mm.; in women 11.5 and 4 mm. respectively. Without entering into a detailed statement of his experiments and arguments, he is led to the conclusion, that the greater width of the glottis during life is the result of a permanent activity of the abductor muscles, due to tonic, rhythmic impulses, received by their ganglia centers in the medulla from the neighboring respiratory center through centripetal fibers of the pneumogastric. He calls this physiologic action of the abductor muscles the abductor tonus.

I cannot conclude my introductory remarks without calling to your attention and emphasizing the far-reaching importance of the proclivity of the abductor muscles to paralysis in progressive organic lesions.

Whilst Rosenbach,[15] on the strength of a case of cancer of the esophagus, accompanied in the beginning by paralysis of both abductors, later of both recurrent nerves, was the first to proclaim that the abductors are the first to suffer from compression of the recurrent, the credit belongs to Semon of proving that this important pathologic condition is seen not only in local lesions, affecting the nerve along its course, but also in central cerebral as well as spinal diseases of an organic character.

His first publication[16] appeared (1881) in an American journal of high standard, devoted to our specialty, but meeting with a premature death on account of the indifference of the laryngologists at that time, viz., the *Archives of Laryngology*. His statements met with a host of opposition and elicited controversies, sometimes of an acrimonious character and not always of the highest scientific standard. By his incessant labors extending from 1881 to 1897 and by publications too numerous to quote, he had finally the satisfaction of having the so-called Semon law universally acknowledged.

It is beyond the scope of these remarks to enumerate the arguments he put forth to support his assertion, the truth of which all of us have had undoubtedly frequent occasions to confirm.

The ultimate, true cause of the proclivity of the abductors to paralysis has not been definitely determined. Grabower[17] sought not long ago to solve the problem by anatomic, instead of experimental research, previously employed. He examined the innervation of the laryngeal muscles in regard to the configuration of their last nerve terminals, and counted the number of nerve fibers in each muscle. He found, that the branch for the abductor does not divide into smaller ones, radiating in different directions, as in the adductors, and that the number of nerve fibers is less in the former than in the latter. The posticus has on the average 281 terminal fibers, the external and internal thyroarytenoid 485, the transversus 279, and the sum of all the adductor fibers is 680.

I have so far not been able to find reports confirming Grabower's investigation, but without feeling competent to pass on its merits, I am free to say that I would consider it a most acceptable solution of the question at issue. It would explain also in a natural way by an anatomic anomaly the first and only exception to Semon's statement during the long period of twenty-five years, observed by Saundby[18] in a case of esophageal cancer, the analysis of which I have to leave to the gentleman treating of peripheral causes.

Semon[19] admits frankly the facts in Saundby's case, but is not able to offer an explanation. On the occasion of his visit to the United States in 1904, I called his attention to Grabower's publication, but the same seemed not to appeal to him. He adheres to his belief in the biologic differences in the structure of the laryngeal muscles and nerves, which is evident from experimentation in young and old animals as well as in different species, and still more from the differential influence of superficial and deep narcosis on the recurrent, which our late member, Hooper,[20] was the first to investigate.

III.—ETIOLOGY.

Recurrent and abductor paralyses of central origin are caused by organic affections, whilst cortical paralysis of a functional character occurs in hysteria, psychic emotions, shock, etc., with concomitant paralysis of the adductors, but not of the recurrent proper.

According to the location and nature of the diseases we can distinguish paralyses due (1) to cortical lesions, (2) to disturbances of the nerve tract in the internal capsule between cortex and bulbus, (3) to bulbar processes, (4) to syphilis, (5) to tumors, exostoses at the cranial base, pressing upon the pneumogastric, (6) to spinal diseases, (7) final paralyses of the Avellis and Hughling Jackson type.

Beginning with paralyses due to cortical lesions, we enter at once a much contested field.

A number of cases have been published in which the authors claim that an unilateral cortical affection had caused an unilateral organic paralysis. Whilst in many instances the history, the laryngeal examination, the postmortem reports, are incomplete or missing, and therefore the proof of the contention made is lacking, there are cases in which the symptoms in the living corresponded with the lesion found after death. Garel,[21] for instance, saw a woman 73 years of age with hemiplegia of the right side and complete paralysis of the left vocal cord. The autopsy showed softening of the left hemisphere and two similar foci in the right, one at the base of the third frontal convolution, to the latter of which Garel attributed the laryngeal paralysis. A microscopic examination of the bulbus had not been made, and consequently its integrity cannot be vouched for.

On the other hand, cases which appeared during life to belong to this class, showed bulbar disease after death. Our member Delavan is quoted amongst the above authors, when relating the history of his patient seen during life,[22] but in his comment on the result of autopsy, he says[23] that his case was a direct proof that paralysis of the larynx of central origin is generally due to bulbar lesion and not to disease of the cortex.

In my opinion there are at the present time extremely few believers in an unilateral laryngeal paralysis brought about by unilateral cerebral disease; otherwise the number of cases would certainly be much larger than we find in the literature, as unilateral cortical lesions, for instance hemorrhage, softening, etc., are not infrequent. Semon in his article in Heymann's Handbook, 1898, could collect only twenty-five cases in which such a coincidence was claimed by the authors. The strongest argument against the occurrence of these paralyses is the anatomic fact that laryngeal movements can be excited by each of the two cortical centers, independent of the loss or destruction of one of them; further, that the experimental extirpation of the cerebral hemispheres, down to the fourth ventricle, does not interfere with the respiratory movements of the cords, the integral function of the abductor.

Affections involving both cortical centers, for instance hemorrhages, are as a rule, not confined to this area alone, and are of such a serious character, that the laryngeal condition is overshadowed by the grave general symptoms. But cases are reported, in which the connection between the cortical and bulbar center through the internal capsule became obliterated by the morbid process with resulting aphasia.

Eisenlohr's[24] patient had general motor disturbances, was absolutely speechless, and the postmorten showed destruction of the posterior third of both thalami optici, partial degeneration of the posterior part of the internal capsule and of the pyramidal tract of the medulla and spinal cord. Pathologic changes of a similar nature were found after death and reported by Pitt[25 26] in a case of chronic nephritis with two apoplectic attacks in quick succession, the second one being followed by loss of voice. In both cases bulbar and cortical lesions were absent and the patients experienced no respiratory difficulties during life.

Similar laryngeal symptoms, associated with lingual and labial pareses are observed in pseudobulbar paralysis, due to disseminated cerebral lesions, in which disease of the medulla and respiratory troubles are absent, and the laryngeal muscles undergo no fatty degeneration.

The subsequent chapters can be considered more briefly, as they deal mostly with well known data, and offer no field for widely dissenting views. Under bulbar paralyses I have included all cerebral diseases, irrespective of the primary seat being in the bulbus or in the hemisphere. Further, the etiologic features of recurrent and abductor paralysis will not be treated of separately, as both are liable to be caused by the same affection, and as the latter is in many instances only a precursor of the former. Prevalence of one or the other type in special diseases will be mentioned in its proper place.

Progressive bulbar paralysis, being due to a degeneration of the motor nuclei in the medulla, represents a genuine type of the bulbar origin of laryngeal paralysis. As a rule paralysis of other organs, tongue, lips, precedes the laryngeal symptoms, which appear sooner or later, dependent on how early the laryngeal nuclei are attacked. The laryngoscopic picture varies in different patients, according to the focus and the intensity of the process. Complete recurrent and unilateral as well as bilateral abductor paralysis has been observed, whilst some authors claim to have seen also adductor paralysis.

Another bulbar affection occupying the same or also adjacent areas, is the apoplectic form of bulbar paralysis, caused by disease of the blood vessels of this region, producing hemorrhages, thrombosis with subsequent softening, also embolism of the vertebral or basilar arteries. Many reported cases of cerebral hemorrhages and encephalomalacia with concomitant laryngeal disturbances are undoubtedly due to such lesions, which have been described already as early as 1879 by Eisenlohr.[27] [28] If the patient survives the attack and its sequelae, we can expect to find a recurrent paralysis, if all the motor fibers are destroyed, otherwise an abductor paralysis.

In the different forms of meningitis we observe anomalies of sensation, but seldom paralysis of the larynx. Our late member Major,[29] saw a patient with bilateral abductor paralysis after cerebro-spinal meningitis; tracheotomy was performed and the patient's condition remained stationary for seven years, when he died from an accident.

Cases of paralysis from multiple cerebro-spinal sclerosis are rare, and the laryngeal symptoms depend on the extent of the disease. Loeri,[30] reported several cases, one of which had recurrent paralysis, J. Solis-Cohen[31] (1886) a case of unilateral, later bilateral abductor paralysis, shown to this association seven years before. From the carefully made autopsy he concludes, that the paralysis has been caused by an ascending sclerosis. Rethi[32] collected in a recent publication 42 cases of sclerosis from the literature and gave a full description of the subject in all its bearings.

Although infectious and toxic paralyses do not belong to the part assigned to me, I cannot refrain from mentioning syphilis of the brain as a cause of recurrent or abductor paralysis. M. Mackenzie[33] published already in 1873 one case, and Lefferts,[34] 1878, two cases of bilateral abductor paralysis due to syphilis, all three cured by specific treatment.

Tumors at the base of the brain are liable to produce paralysis by pressure on the pneumogastric. The symptoms are generally not confined to the larynx alone, as the extension of the growth is apt to implicate other nerve roots also. Nothnagel[35] communicated an exceedingly interesting case of an extended unilateral paraylsis, involving the recurrent of the same side, which was due to pressure from a large abscess at the cranial base, found at the autopsy. Similar symptoms we must be prepared to meet in affections of the cranial bones, exostoses, fractures of the base of the skull, etc.

The importance and the deleterious influence of locomotor ataxia on the larynx is too well known to all of us to need but brief remarks. General medicine is indebted to laryngology because in more than one instance the laryngologist detected abductor paralysis in an otherwise healthy person with a perfectly clear voice, and suspicion being aroused, the examination of the patient revealed locomotor ataxia. By far the most prevalent type is abductor paralysis, of which again the bilateral form is more frequent than in any other disease. Burger[36] collected in his excellent monograph 71 cases of paralysis in tabetics, covering the period from 1866 to 1891, forty-three of which had abductor paralysis. Still more striking is Sendziak's[37] supplementary table of 53 cases of paralysis published between 1892 and 1898. Of these six only had recurrent, of the balance 22 unilateral and 25 bilateral abductor paralysis.

In the remaining spinal diseases we find only isolated cases of laryngeal paralysis, a few of which may be mentioned. Loeri[38] observed a case of bilateral recurrent paralysis in amyotrophic lateral sclerosis, and one in acute spinal leptomeningitis, Schroetter[39] and Gavello,[40] each a case of unilateral recurrent paralysis in syringomyelia, and Koschlakoff,[41] a bilateral abductor paralysis in progressive muscular atrophy.

Although finding scant consideration in the text books, a series of unilateral laryngeal paralyses, associated with such of other organs is of sufficient interest to be mentioned.

Avellis[42] was the first to draw attention to the occurrence of simultaneous unilateral paralysis of the larynx and soft palate—the genuine Avellis-syndrome. In another form the muscles of the neck are also paralyzed. If hemiplegia of the larynx, pharynx and tongue exists, we have the Hughling-Jackson type. A typical case of the latter with abductor paralysis was demonstrated in the London laryngological society by Semon,[43] who in consideration of the general symptoms, believed it to be of central origin, due to a meningeal thickening at the base of the brain. Tapia,[44] who observed unilateral paralysis of the larynx and tongue, called it the fourth type, and finally Pohli[45] gave a complete description of the subject in the *Italian Archives,* 1906.

BIBLIOGRAPHY.

1. Die Krankheiten des Kehlkopfes, 1901, p. 13.
2. Archiv für Anatomie und Physiologie, 1884, Vol. Physiology, p. 203.

3. Phil. Transact. Royal Society of London, Vol. 181, 1890, pp. 187-211.
4. International Medical Congress, 1890, Vol. iv, Part 12, p. 132.
5. British Medical Journal, Aug. 24, 1895, p. 481.
6. Experimentelle Untersuchungen über die Funktion der Nerven und Muskeln des Kehlkopfes, Würzburg, 1873.
7. Archiv für Laryngologie, Vol. xii, 1902, p. 70.
8. Ibidem, Vol. ii, 1895, p. 144.
9. Ibidem, Vol. x, 1900, p. 320.
10. Lehrbuch der Nerven Krankheiten, 4 Auflage, 1st. Band, p. 511.
11. Proceedings of the Royal Society, Vol. 51, 1892, March 31st, p. 102.
12. Bolletino d. Mal. dell' Orecchio, July, 1893, p. 151.
13. Archiv für Anatomie und Physiologie, 1905, Heft 3 and 4, p. 396.
14. Proceedings of the Royal Society, Vol. 48, 1890, p. 403.
15. Breslauer aerztliche Zeitung, 1880, Nos. 2 and 3.
16. Archives of Laryngology, Vol. ii, 1881, No. 3, p. 197.
17. Archiv für Laryngologie, Vol. xvi, 1904, p. 189.
18. British Medical Journal, 1904, March 12th.
19. Internationales Centralblatt für Laryngologie, 1904, p. 225.
20. Transactions American Laryngological Association, 1887, p. 41; 1888, p. 163.
21. Annales des maladies d'oreille, etc., 1886, p. 218.
22. Transactions American Laryngological Association, 1884, p. 82.
23. Ibidem, 1888, p. 195.
24. Deutsche Zeitschrift für Nervenheilkunde, Vol. i, 1891, p. 388.
25. British Medical Journal, 1893, May 20, p. 1058.
26. Transactions Clinical Society, London, Vol. xxvi, 1893, p. 250.
27. Archiv für Psychiatrie, Vol. ix, 1879, cases 3 and 4, pp. 19 and 22.
28. Ibidem, Vol. xix, 1888, p. 316, containing also report of autopsy of cases 3 and 4 of 1879, pp. 315 and 316.
29. N. Y. Medical Journal, 1890, June 28.
30. Die durch anderweitige Erkrankungen bedingten Veränderungen des Rachens, Kehlkopfs und der Luftribe, 1885, p. 13.
31. Transactions Am. Laryng. Ass'n, 1886, p. 42.
32. Die laryngealen Erscheinungen bei multipler Sclerose des Gehirns und Rückenmarks, Wein, 1907.
33. Diseases of the Nose and Throat, London, 1880, Vol. i, p. 429, case 6.
34. N. Y. Medical Journal, 1878, December.
35. Wiener medizinische Blätter, 1884, No. 9, February 28, p. 256.
36. Die laryngealen Stoerungen der Tabes dorsalis, Leyden, 1891.
37. Klinische Vortraege aus dem Gebiete der Otologie und Pharyngo-Rhinologie, 1901, Vol. iv, No. 3, pp. 170-171.
38. Loco citato, pp. 16 and 19.
39. Krankheiten des Kehlkopfs, Wien, 1892, p. 413.
40. Arch. Ital. di Otol., Rinol. e Laring., Vol. xvii, 1905, November, p. 25.

41. Internationales Centralblatt für Laryngologie, Vol. ii, 1885-6, p. 229.
42. Berliner Klinik, 1891, Vol. 40, October.
43. Journal of Laryngology, 1907, February, p. 50.
44. Annales des maladies d'oreille, August, 1906, p. 134.
45. Arch. Ital. di Otologia, etc., Vol. xvii, 1906, March, p. 215.

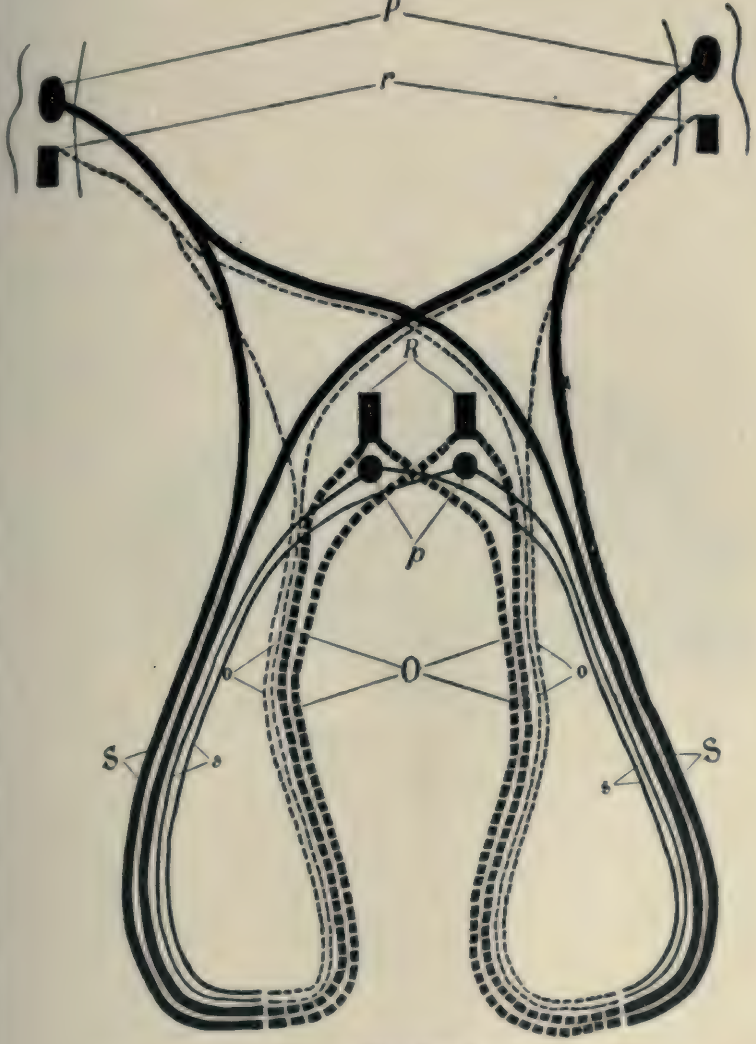

DIAGRAM OF
Phonation and **Respiration** in **Cortical** P-r and
Bulbar p-R Centre with tracts in internal
Capsule : full Lines for Adductors ▬ S-s
broken Lines for Abductors == O-o

FIGURE 1.

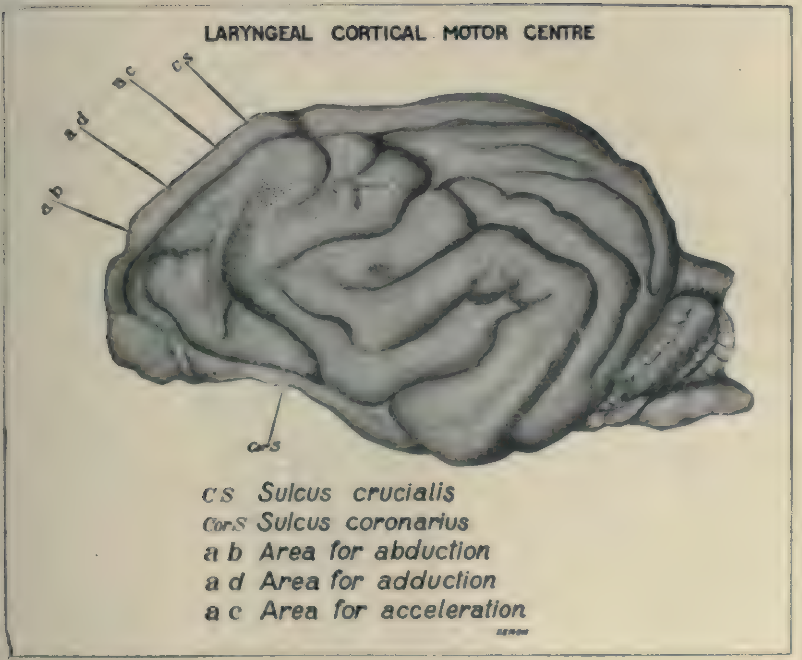
LARYNGEAL CORTICAL MOTOR CENTRE
c s
a c
a d
a b
Cor S
c s Sulcus crucialis
Cor S Sulcus coronarius
a b Area for abduction
a d Area for adduction
a c Area for acceleration

FIGURE 2.

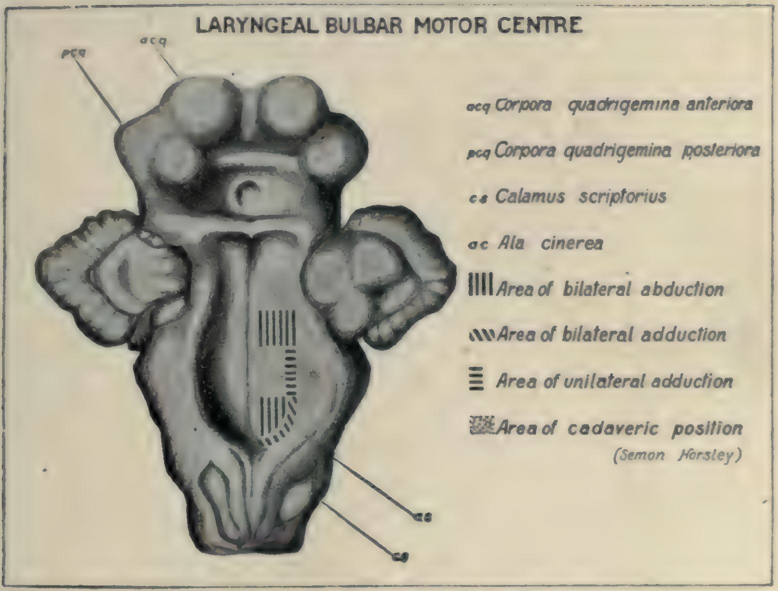
LARYNGEAL BULBAR MOTOR CENTRE
pcq
acq
acq Corpora quadrigemina anteriora
pcq Corpora quadrigemina posteriora
cs Calamus scriptorius
ac Ala cinerea
Area of bilateral abduction
Area of bilateral adduction
Area of unilateral adduction
Area of cadaveric position
(Semon Horsley)
ac
cs

FIGURE 3.

NUCLEI OF CEREBRAL NERVES

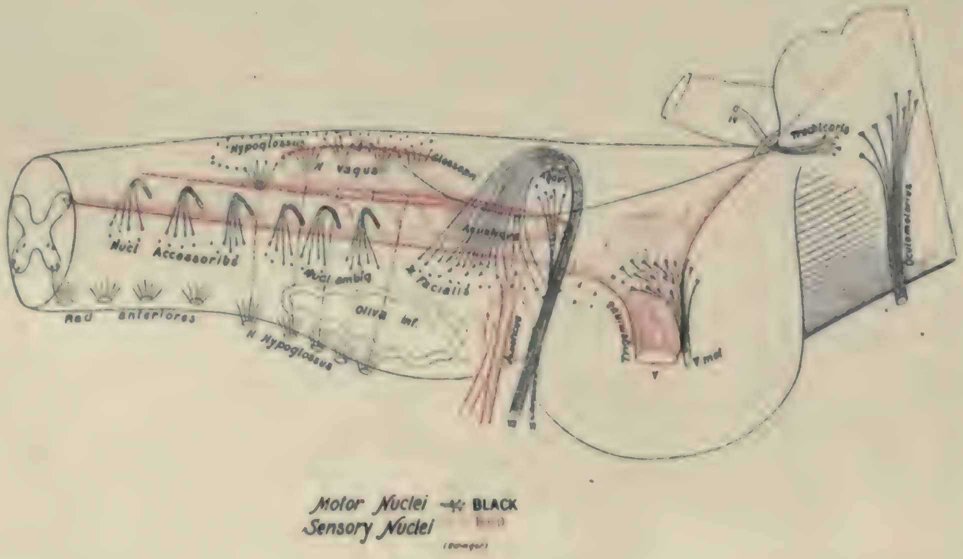

FIGURE 4.

XXII.

RECURRENT AND ABDUCTOR PARALYSIS OF THE LARYNX. DIAGNOSIS AND TREATMENT.*

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In the diagnosis of unilateral recurrent nerve paralysis, which when complete results in the fixation of one vocal cord in the cadaveric position, the cracked, softly raucous voice, is suggestive rather than distinctive, but it is usually the one small symptom of an inversely grave condition which first causes the patient to seek advice. It is nature's distress signal, full of meaning to him who reads the code. The same is true of that partial stridor which may or may not usher in an unilateral abductor paralysis in which the dilator muscle alone being powerless, the vocal cord is retained in a position closing over one-half of the lumen of the larynx. Either symptom demands a prompt resort to laryngoscopic inspection, but even then by reason of the fact that patients are liable to present themselves for the first time during "a cold" or other aggravating influence, confusing departures from the typical image may be encountered, and one turns next to the really vital phase of the diagnosis; a search of the chest, neck and central nervous system, for the cause and significance of the vocal paralysis. The tortuous course and complex composition of the recurrent nerves should be kept well in mind. Branching from the pneumogastric, the left one takes a turn under the arch of the aorta, *between it and the left bronchus,* upward to the larynx, while the one on the right passes under the subclavian artery; both being liable to compression by aneurysm, mediastinal and cervical tumors and, on the right, by disease at the apex of the lung or its pleura, which is crossed by the nerve. The similarity of lettering of the two words abduction and adduction is unfortunate, for in addition to typographic errors, a conscious mental effort is necessary to avoid confusing one with the other, but as applied to the movements of the vocal cords, they are more exact in meaning than any other available terms. The opposing muscles which effect the to and fro motion of the vocal cords, adduction for vocal vibration, abduction for breathing space, are innervated by different fibers, which merely travel in the same recurrent nerve trunk, either set of fibers alone to any degree or both sets together, being subject to paralysis. Semon's law refers to the fact that the abductor fibers and muscles are the more vulnerable of the two, yielding first to an equal pressure on the trunk, and in consequence every complete paralysis of a vocal cord by pressure must have passed through a preliminary stage, however brief, of abductor paralysis alone.

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*Read as part of a Symposium before the American Laryngological Association, May 11, 1908.

ANEURYSM GROUP.

The typical image of complete unilateral recurrent nerve paralysis illustrated in Fig. 1, is introduced as a standard from which to describe the diagnostic variations. It represents the larynx of a middle-aged salesman, whose voice, he said, "Failed all at once, one day, while selling goods." Not only is the left vocal cord motionless in the cadaveric position, but it is relaxed, tends to bay outward, and its arytenoid, which is *not swollen,* has "fallen in," i. e., collapsed forward, which shortens the vocal cord and permits the cord to sink to a plane slightly below the level of its fellow. On phonation, the left arytenoid may give a twitch, but fails to straighten up or convey more than a mere tremor to the cord, while the opposite arytenoid and vocal cord, in an effort to compensate, overreach the center line, the sound arytenoid crossing a trifle usually in front or possibly behind the paralytic one and the healthy cord coming nearly but not quite over to the paralyzed one. These are not insignificant details for their presence collectively, rather than the cadaveric position alone of the cord, insures the fact of a complete recurrent nerve paralysis. Its cause in this instance was equally evident. There was substernal pain and a radial pulsus differens, but no other symptom, excepting that in the fluoroscope, a small pulsating aneurysm of the arch of the aorta was visible. Five months later, while walking on the street, this patient fell dead with a gush of blood from the mouth—the aneurysm had burst; presumably into the left bronchus.

Life's duration in aneurysm, however, is seldom so brief. Dating from the early period of vocal paralysis, it has often surpassed my expectation; in one 10 years, in another 5, while others are now living at 2, 3 and 4½ years. The seemingly prolonged course, last mentioned, the patient being still active, is explained by the fact that it dates from the earliest demonstrable stage in the development of the aneurysm, as evidenced by the presence of unilateral abductor paralysis, which is the first definite effect of pressure on the recurrent laryngeal nerve. A sketch of the larynx made at that time (Fig. 2), represents the typical image of unilateral abductor paralysis and is intended to show not only the vocal cord motionless in the median or closed position, but also that the arytenoid has not yet "fallen in," hence the cord is but little depressed or shortened and not very much relaxed. Paralysis limited to abduction is not the only possible interpretation of the median position of a vocal cord, for one of the most deceptive of the variations from the typical image of complete recurrent paralysis is a fixation of the cord, not exactly in, but quite near to the median line instead of in the usual cadaveric position. Either form of paralysis is suggestive of an aneurysm, but with paralysis of the abductor fibers alone, indicating an earlier stage and more limited development of the aneurysm, a longer course is to be expected and an "arrest" or even a retrogression might not be impossible. Hence, in confirmation of the memorandum and sketch made of abductor paralysis in this case I will quote the additional notation inscribed that, "The vocal cord was not much relaxed and that the arytenoid, although standing a little forward, had not yet 'fallen in,'" a combination of conditions which I interpret to indicate an incomplete degree of paralysis of the recurrent nerve which, in connection with the median position of the cord, could be no other than an abductor paralysis. His x-ray print I find somewhat blurred by the effects of time, still it distinctly shows, representing the aortic arch, a broadened shadow which rises in the back above the normal limit of the fourth rib, with a bulge to the left which centers near the second rib, and a smaller bulge to the right at the fourth intercostal space. A fluoroscopic examination also, by Dr. John Kales and the author, was recorded at that time as follows: "A distinct but small pulsating bulge in the descending part of the arch of the aorta could be plainly seen."

Since writing the above description with its deductions relative to the 4½ years' duration of an aortic aneurysm, this patient, known to have remained vigorous so long as to suggest a shade of doubt in the diagnosis, has returned, thus enabling me to complete the evidence to date with respect both to the laryngeal paralysis of which he is conscious only when further disabled by a "cold" as at present, and to the aneurysm which does not even yet cause any subjective symptoms other than the vocal cord paralysis. Re-examination confirms all of the original findings, with natural progression. The position of the left vocal cord has advanced from the typical abductor or first position, partly but not wholly, into that of complete recurrent paralysis. It now exemplifies that variation from the typical image of complete recurrent paralysis described as "a fixation of the cord not exactly in, but quite near to the median line instead of in the usual cadaveric position." Intensified tubular breathing, the physical sign emphasized as coming next after vocal paralysis, is now heard in the back at the left, above the angle of the scapula and, as might be expected with compression of a bronchial tube, the vocal resonance and fremitus are diminished at the same point. A blurred, widely transmitted first sound stands for the aneurysmal bruit and the subclavian impulse is in excess of the natural. The fluoroscope and x-ray plate show a clearly defined sacculated aneurysm with a diffused distension of the rest of the aortic arch.

It requires but a slight expansion of the aorta at a given spot to compress the left recurrent nerve, hence the frequency of even a complete recurrent paralysis at comparatively an early period, when perchance one may search in vain for any other classical sign of the aneurysm, which in fact can still be so small that even a radiograph will leave a doubt behind, as in Fig. 3. Here there was neither bruit, thrill, tracheal tug nor protrusion, yet there was paralysis of the left vocal cord and there was, moreover, a noteworthy accessory physical sign, i. e., an exaggerated bronchial breathing transmitted to the back, a symptom which coming early, pari passu with vocal paralysis, should be of special disagnostic value to the laryngologist, for an aneurysm which compresses this nerve must at its very next step, compress the left bronchus. An appreciable diminution in vesicular expansion comes only later.

It has been noted that in the typical image of complete paralysis, the "fallen in" arytenoid is not swollen. but exceptionally, as a departure from the typical image, a coexisting swelling of the arytenoid is found which is liable to disguise the paralytic aspect and to cause the condition to simulate a crico-arytenoid ankylosis with fixation of the cord. Fig. 4 represents this important variation. When first observed, the right cord was stationary, but in a doubtful position, the arytenoid and subglottic tissue was swollen and the left cord, also, was sluggish in movement. It was only after some weeks of detergent local treatment that the cadaveric position of the right cord could be accurately determined, the left cord, meanwhile, having definitely weakened into the first position, that of abductor paralysis, which in conjunction with the other lesions sufficed to cause an alarming respiratory stridor on exertion or on "taking cold." Aneurysm was now evidenced by other .signs and its existence was later verified in Berlin by Dr. Krause, who, in explanation of the swollen arytenoid, wrote: "The small tumefaction of the arytenoid is a little bit unclear, but may be caused by a prolapsus of the cartilage." Hooper,[1] also, once drew our attention to certain "anomalous muscular fasiculi,[2] innervated from other sources, which when present in conjunction with recurrent paralysis would serve to pull the arytenoid forcibly forward." The possibility thus, of a partial dislocation of the arytenoid amounting to a prolapse instead of merely a collapse, is a striking point in diagnosis to be remembered, especially with reference to the differentiation of paralysis from ankylosis, which is rendered thereby all the more difficult.

The bilateral inflammatory form of paresis of the internal tensors with its familiar ellipse, is in no danger of being mistaken for recurrent nerve paralysis, but I have twice observed in clergymen an unilateral internal and external tensor paralysis, induced by vocal strain, but of so severe a grade as to simulate and perhaps in a way to partake of a recurrent nerve paralysis. Assuming for it a neuromuscular rather than a strictly myopathic basis, it is the nearest approach to a direct impairment of the nerve endings which my series affords. The level of the cord is lowered, its range of movement to and fro lessened, and the arytenoid disposed to collapse, but it is distinguishable by a predominance of the flaccid tensor element, absence of lead, alcohol or other toxemias, absence of any cause of pressure, and it finally recovers by rest to the voice.

Out of a total of forty cases upon which this study is based, the left recurrent nerve was paralyzed by an aneurysm in eight and the right recurrent in six, including in both numbers those in whom the opposite cord was likewise but less affected. Concerning the treatment, in only a few of the aneurysm group was local medication indicated and then not for the paralysis, itself, but rather to aid in strengthening the voice or in diminishing the stridor by controlling the inflammatory complications. Moderation in voice use is enjoined on all and soon has its reward in distinctly improved tones, the voice, in time, through compensation, often becoming fairly good for conversational use. The prognosis of aneurysm, in general, is not so absolutely bad, and discovered at an early stage it should be better. Syphilitic arteritis in other situations, for example, in the brain, elicits and yields to a vigorous specific treatment; why not so in an early stage of aortic arteritis?

PLEURITIS GROUP.

Four additional paralyses of the right cord compose the pleuritis group. All were tuberculous and in none could any other cause be assigned than an assumed circumscribed pleuritis. In one of them, whatever might have been the exact cause of a recent and complete unilateral right recurrent paralysis, which showed a typical image, a perfect restoration of power to the cord, was gradually attained contemporaneously with a treatment by fresh air and rest for the incipient tuberculosis. Our means of certifying a diagnosis in this group are as yet sparse. The x-ray should be of service for the purpose of excluding or establishing the presence of enlarged mediastinal lymphatic glands on the left side, which in tuberculous subjects constitute a possible cause of pressure on the recurrent nerves; but on the right side, enlarged glands so located would scarcely be beyond the reach of deep palpation. However, a radiograph was taken of this particular patient, who recovered, and no abnormality was shown. Her reaction to the tuberculin test was positive.

TUMOR GROUP.

Another departure from the typical image of complete paralysis is presented in Fig. 5, in that the ventricular band is hypertrophied and almost covers over the paralyzed cord, nature having gradually put forward this false cord in compensation; it vibrates with the sound cord and produces a fair voice. It is selected from my next group of eight cases, in each of which a tumor was the cause, and in all but two of which, unlike in the aneurysm group, the vocal disability appeared only as a late and gradual development. In this patient a primary malignant growth had been removed from the chest wall three years previously, a fact which he considered so irrelevant to vocal trouble that he deliberately falsified when I asked about the scar, saying he had been struck by a shell during the war. A grimly humorous episode served, in the interests of science, to complete my record of this case, for the autopsy having been held in my absence, the widow, who it appears had fervently disapproved of his falsehood about the scar, entered one day and dramatically deposited a large package before me on the desk, stating exultingly, "There, that's what he had." It proved to be a sarcomatous tumor from the mediastinum.

In this group also I find another observation, that late in the course of a carcinoma of the esophagus, an irritable cough and weak voice having appeared within a fortnight, the left cord was found motionless, near to, but not exactly at the line of abductor paralysis and that the arytenoid had not "fallen in." Evidently the paralysis was progressing gradually and a remnant of innervation remained.

Esophagoscopy has been helpful, although usually, by the time a vocal paralysis supervenes in carcinoma of the esophagus, the diagnosis is plain. I was once temporarily misled with respect to the larynx by a condition which suggested a dawning vocal paralysis in consequence of esophageal neoplasm. There was post-prandial eructation of what the patient called "a lot of phlegm," but which in reality was a fermented mixture of mucus and food which irritated the arytenoids and enfeebled the voice, but obscured the image. The x-ray disclosed a bulge, and showed the arrested bougie within it (Fig. 6). The esophagoscope, passed under general anesthesia, was arrested at this same point where an easily bleeding mammillated mucosa was plainly seen, the exact nature of which, however, it was impossible to interpret. As a palliative treatment in this particular case, which proved not to be one of paralysis, the expedient of assuming an inverted recumbent position after each meal, which by gravity emptied the pouch, has afforded a gratifying relief to the throat symptoms, which two years ago were quite distressing. Meanwhile, the obstruction in the esophagus, whatever its nature, remains still in statu quo. Having the appearance of a neoplasm, it is of interest in this connection because it is differentiated from aneurysm by the fact that it does not pulsate as observed in the fluoroscope, and because it is in line with the dictum that vocal parlysis, unlike in aneurysm, is not an early symptom of neoplasm of the esophagus unless the latter is located high up.

We recognize in the so-called "lazy cord" a symptom of carcinoma of the larynx, but I think it is little known that total immobility of a cord in the cadaveric position may be the first visible evidence of carcinoma of the larynx. Through a surprise of this sort at autopsy, I am able to duplicate the single observation of it which Semon[3] records in these words: "Einmal habe ich eine bösartige Kehlkopfneubildung in Form der Unbeweglichkeit einer Stimmlippe in Cadaverstellung beginnen sehen."

The other exception to a late development in the tumor group occurred with a high carcinoma of the esophagus and involved both cords, the left a cadaveric, the right an abductor paralysis. The end was then approaching, but aphonia two years before had been the very first symptom, the reason being that high up in the neck the recurrent nerves lie so close to the esophagus that not only one but both nerves are liable to be affected by pressure or to be incorporated in the carcinoma almost at the start.

Goitre was responsible but once for a complete paralysis of a vocal cord. It was a small, firm enlargement of the left lobe, which was treated by surgical removal, but the nerve had already atrophied. In goitre, when the voice begins to weaken, the cords should receive the closest scrutiny, for surely the first definite evidence of paralysis would be an additional incentive to surgical interference. This principle pertains equally to other cervical tumors and to enlarged lymphatic glands. Its value was brilliantly evidenced in one of this group by a complete restoration of voice which followed the excision of three tuberculous glands in a school teacher whose left vocal cord previously had been motionless in the cadaveric position.

The large variety of conditions capable of inducing recurrent nerve paralysis, is emphasized by an error in diagnosis of which I became apprised only when tracing the patients for the purpose of this report. The image of complete paralysis of the left cord was typical and without any tumefaction inside the throat. A swelling in the neck at the site of the thyroid gland was naturally mistaken for a goitre, the subject being a girl of 18 years. Goitre was accepted as the cause of the paralysis and its surgical removal recommended, but not done. Signs of tuberculosis at the right pulmonary apex seemed to explain her anaemic or cachectic appearance. The supposed goitre turned out to be a malignant neoplasm, which terminated in the usual manner about a year later.

CENTRIC BULBAR ABDUCTOR GROUP.

Like other respiratory muscles, the abductors of the vocal cords (dilators) being impelled by reflex action, have their main centers in the medulla oblongata (bulbus), so near together that naturally the symmetrical double abductor paralyses predominate in my bulbar group. The mere fact of symmetry in persistent abductor paralysis is indicative of a bulbar lesion. When uncomplicated the appearance in the mirror is pathognomonic. The flaccid cords which will not separate, their edges flapping up and down like a sail in the wind, are unmistakable. The question of a possible overpowering spasm of the opposing adductor muscles did not need consideration in any one of this class, although it may arise especially in connection with the intermittent stridor of an incipient stage of abductor paralysis and also in the unilateral type. The differentiation from ankylosis may require the same degree of critical observation as recurrent nerve paralysis. As a paralytic immobility is liable to lead into a sort of ankylosis or stiffness of the joints, and moreover there is at least one form of arytenoid arthritis, previously described by me under the name of arthritis deformans of the larynx,[4] which causes a *symmetrical* ankylosis without much tumefaction.

An energetic course of mercurial and iodid medication is indicated whenever the centric degeneration is connected with tabes or with any other suspicion, however remote, of syphilis, for exceptionally one finds a practical recovery recorded. Eight cases of abductor paralysis compose this bulbar group, four of them being of the persistent symmetrical double type. Unfortunately, in all of these four the varied treatment did naught but eventuate in tracheotomy which, although far from an ideal remedy, remains still the best life-saving device. For eight years one of them had been wearing a tracheal tube of the sort which is fitted with a ball and socket valve which enabled him not only to inspire with freedom, but to talk with ease, for the obstruction by abductor paralysis is inspiratory only, the downward air pressure forcing the vocal cords even closer together, while the outward air pressure balloons them slightly apart. Thus, with the tube in place, by a skillfully modulated use of the expiratory current which served momentarily to close the tubal valve, second to divert the air from the tube through the chink of his glottis, he could uninterruptedly intone and articulate sentences of considerable length. Neither this patient nor another, who had been dependent for his life, during several years, upon a simple tube which in order to effect conversation required its stoppage by the usual conspicuous method of holding the finger at its opening in the neck, would, on account of the risk to speech involved, consider any effort to widen the glottis to the small extent which would be adequate for ordinary breathing, either by means of an excision of one cord or by the proposed method of deliberately causing it to assume the cadaveric position by intentionally cutting the recurrent nerve. The latter method, supplemented perhaps by mobilization of the cryco-arytenoid joints if found stiffened, would seem worthy of a trial, considering that prolonged dependence upon the wearing of a tracheal tube is in itself a menace to life. Neither of them suffered from tabes, but both gave a history of syphilis.

Not only the parasyphilitic degenerations, but also the degeneration incidental to advanced age may happen to strike these minute centers in the medulla. It was the only apparent lesion, without other cause, in a woman of 75 years, in whom a treatment by emollients and mild vascular astringents sufficed, under careful observation, to defer for some months, the ultimate tracheotomy. The same line of treatment in conjunction with antisyphilitic remedies was finally successful in another one of this group, but of a different type, the condition being a partial and unsymmetrical abductor paralysis in which the stridor became serious only during periods of acute exacerbation by "a cold."

The remaining representation in the bulbar group is of that combination which anatomically is far from clear, now that the spinal accessory nerve is not considered contributory to the recurrent nerve, in that the abductor paralysis being unilateral and devoid of symptoms was discovered incidentally, as the complaints had reference to difficulty in swallowing and articulating, in consequence of paralysis of the palatal, pharyngeal and lingual muscles together with atrophy, in one instance, of the trapezius and sternocleidomastoid muscles. One, the victim of a generalized arteriosclerosis without syphilis, was not responsive to any form of treatment and succumbed from inanition, while another, a syphilitic, was benefited by energetic specific medication to the extent of regaining an ability to swallow liquids without a nasal reflux and to articulate with fair distinctness.

CENTRIC CORTICAL GROUP.

Owing to the bilateral laryngeal action which ensues from an unilateral cortical stimulus, it is supposed that paralysis of one vocal cord from a lesion of the opposite side of the brain, really does not occur and that some fallacy has underlain the few apparent cases which have been recorded.[5] Delavan[6] reports in convincing detail one case previously published as cortical, in which at autopsy was found a conjoined bulbar disease; but probably the fallacy also lies in mistaking the "stroke" of a syphilitic endarteritis for that of true hemorrhagic apoplexy. Laryngeal paresis can, of course, ensue from organic cortical lesions which affect the brain centers of both sides or interrupt the nerve fibers in transit from both sides, as in syphilitic degenerations or arteriosclerosis; and the six cases which compose my centric cortical group may have been either of this nature or else similar to Delavan's case. In three of them, the vocal paralysis occurred in connection with attacks of so-called apoplexy, but as they were men under 50 years of age, with venereal histories, did not lose consciousness at the time of the "stroke" and had recovered in some measure, the attacks doubtless were due not to unilateral hemorrhage, but to syphilitic cerebral arterio-thrombosis which is often unequally bilateral. In none, was a typical image of paralysis of any particular group of laryngeal muscles presented, although the left cord in one was nearly, but not quite, motionless in the cadaveric position, the condition in two being an incomplete bilateral paresis which predominated in the larynx on the side of the paretic arm and leg, as evidenced by a feeble closure of the flaccid cords in a slanting position. Either an enfeeblement of the pharyngeal muscles, or impairment of articulation, or mild aphasia, was associated with the laryngeal paresis in one or another of this subgroup. One of them has remained under observation, receiving antisyphilitic treatment during a period of seven years and is now well.

The remaining three patients of this group were persons somewhat advanced in years who presented symptoms, in general, of cerebral degeneration. the paresis of the vocal cords being the cause only in part of the difficulty in speaking, although hoarseness and weakness of the voice were particular complaints. The laryngeal image was not typical and represented an unequal paresis of both cords rather than a complete paralysis of either cord. The local treatment was mildly palliative only, but proved far from useless, as the detergents and emollients applied by simple methods served to lessen the friction of vocalization and thus represented to the aged sufferers the difference between misery and contentment.

REFERENCES.

1. Franklin H. Hooper. Transactions of the American Laryngological Association, 1886, p. 25.
2. Thyro-arytenoidei superiores muscles.
3. Felix Semon. Heymann's Handbuch, vol. i, p. 744.
4. Casselberry. Transactions of the American Laryngological Association, 1893, p. 18.
5. Lewin. Berl. klin. Wochenschrift, 1874, p. 10. Cited by Semon.
6. Delavan. Transactions American Laryngological Association, 1884, p. 82, and 1888, p. 195.

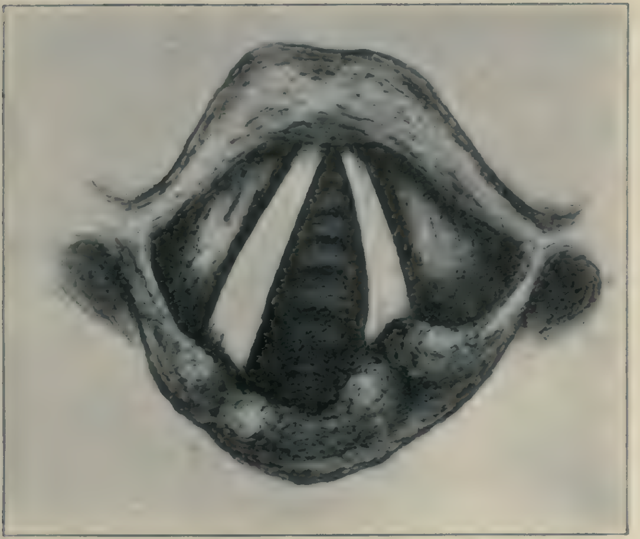

FIG. 1. The typical image of complete left recurrent paralysis. The vocal cord, in the cadaveric position, appears relaxed, narrowed, shortened and depressed; the arytenoid, "fallen in," not swollen.

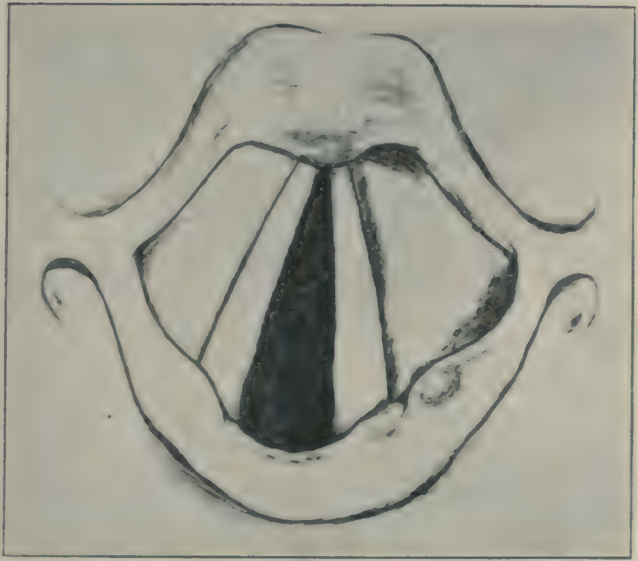

FIG. 2. The typical image of unilateral abductor paralysis. The vocal cord in the median or closed position. Arytenoid only slightly "fallen in," hence the cord is but little depressed or shortened.

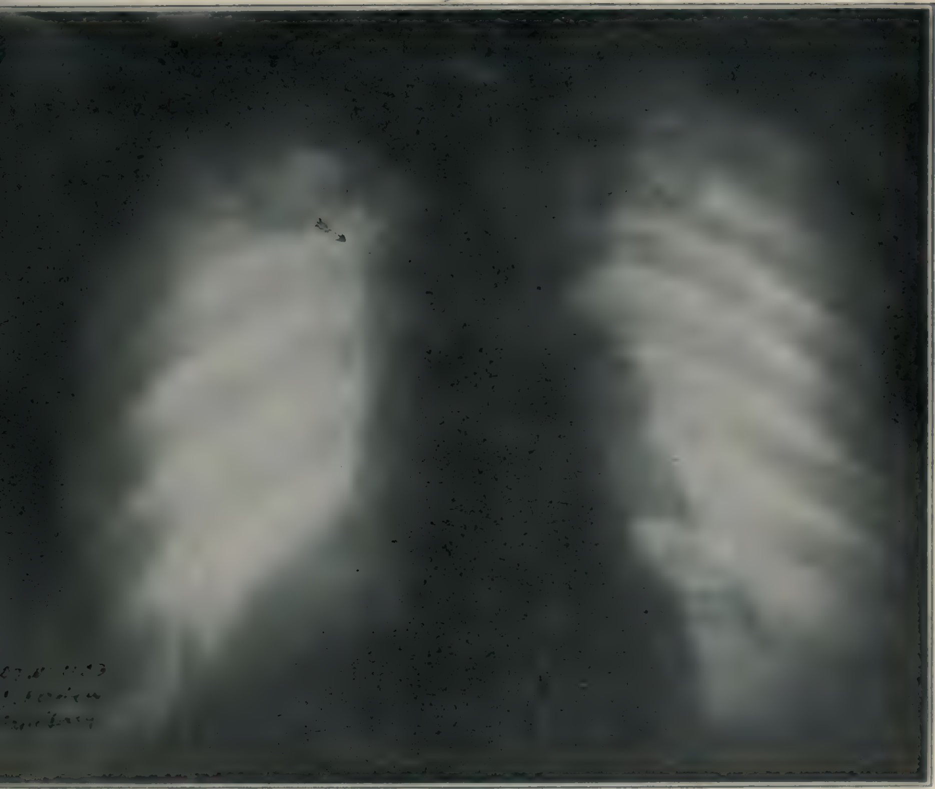

FIG. 3. Aneurysm, so small that the radiograph alone would have left it doubtful. Complete paralysis of the left vocal cord and exaggerated bronchial breathing present.

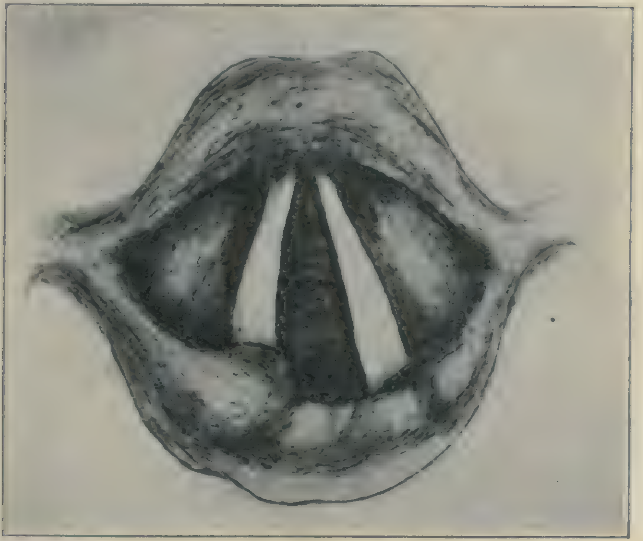

FIG. 4. A variation from the typical image of recurrent paralysis. The arytenoid is swollen.

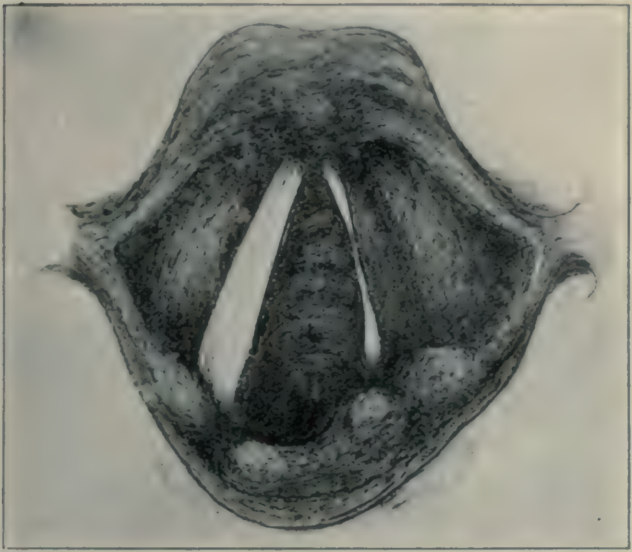

FIG. 5. A variation from the typical image of recurrent paralysis. The ventricular band is enlarged.

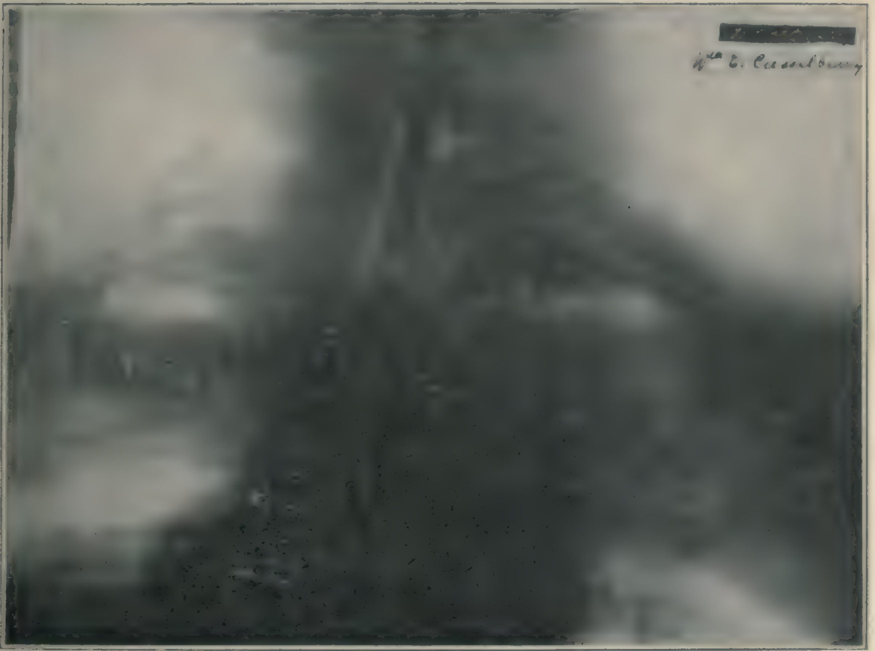

FIG. 6. Esophageal neoplasm. Differentiated from aneurysm by absence of pulsation when observed in the fluoroscope.

XXIII.

THE ETIOLOGY OF PARALYSIS OF THE RECURRENT LARYNGEAL NERVES OF PERIPHERAL ORIGIN.*

By D. Bryson Delavan, M. D.,

New York.

Reviewing the history of this subject, we find that at a period centering about twenty years ago, much interest was shown in its study. Led by Prof. Hermann Krause in Europe and by the late Prof. Franklin H. Hooper in this country, excellent laboratory work was done and some important facts were demonstrated. Of the main questions at issue, however, many were unanswered, in spite of the earnest efforts of a number of well qualified investigators and of controversies which in some instances assumed the proportions of bitter feuds. Of late years not much has been advanced on the subject and that little has not been illuminating. Possibly the difficulty of the study and the lack of success attending its investigation have discouraged progress. Possibly again in view of the present popularity of other adjacent fields, the larynx has received less attention recently than it deserves.

The serious nature of recurrent paralysis and the not inconsiderable number of patients affected by it should certainly stimulate us to more earnest effort towards the better understanding and explanation of the causes which bring it about.

In many varieties of the affection the cause is clear. In others it is obscure, as witness the number of cases in which the diagnosis has either not been made or has been clouded by conflicting opinions as to the actual effect of the supposed cause.

I shall be able to do little more than call attention to some of the questions which the subject suggests and ask you to interest yourselves in taking up their investigation.

Indeed, in spite of all that has been written on the subject, it may be said that the study of this department is in its infancy, for it has not even been determined whether certain toxic paralyses of the recurrents are due to central causes, to changes in the nerve elements or to neuritis from severe surface irritation.

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*Read before the American Laryngological Association, thirtieth annual meeting, Montreal, May 11, 1908.

Recurrent paralysis may be either unilateral or bilateral, and only the posterior fibers of the nerve may be involved, as in abductor paralysis, or, the paralysis may be complete.

Shurly[1] quotes Avellis to the effect that of 150 cases, the cause was discovered in only 85. Of the above 150 cases, plus 19 collected by Sendziak,[2] 113 were males and 50 were females. In the same series of cases, the paralysis was found 46 times on the right side, 92 times on the left side, and 12 times on both sides. In 41 cases of Sendziak, 5 were right, 15 left and 21 bilateral. Unfortunately little reliability can be based upon these statistics. Three points become evident from them, however, as has been suggested by Lermoyez, namely:

The preponderance of peripheral paralysis over that caused by central lesions; the excess of cases in which the paralysis has been on the left side, and, finally, the greater liability of recurrent paralysis among men.

As to age, of 19 cases, 15 occurred between the 20th and 60th years. The youngest was a child of 2 years, the eldest was 59.

Sendziak claims to have collected 1017 cases, which he has classified according to the form of the paralysis, as follows:

1.	Abductor Paralysis	526		
2.	Paralysis of Recurrent	491		1017
1.	a. Unilateral Abductor Paralysis	283		
	b. Bilateral Abductor Paralysis	243	526	
2.	a. Unilateral Recurrent Paralysis	397		
	b. Bilateral Recurrent Paralysis	94	491	
			1017	1017

Among the earliest observations noted with regard to them was the fact that if the recurrent were injured, dyspnea was a prompt result. This was made apparent through the effect upon them of wounds of the throat and neck. Later on the possibility of paralysis from pressure was recognized.

Finally, within recent years, a few observers have called attention to the occurrence of paralysis from toxic causes.

Many cases have been reported in which the movements of the larynx have been affected by peculiar conditions of the blood due to disease or to the ingestion into the system of certain well-known poisons. Thus gradually it has become apparent that paralysis of the recurrents of peripheral origin may be due to many causes.

The consideration of paralysis due to traumatism and to pressure are very interesting topics. Moreover the subjects are far from having been exhausted. Many things remain to be explained regarding both of them.

Paralysis due to toxic agencies, however, is a subject which has been hitherto little studied. It is replete with problems of many kinds and these again depend upon questions of surpassing interest. Few fresher or more attractive fields could be offered to the investigator, whether physiologist, pathologist or chemist.

In studying the reports of cases by various authors, the writer has been impressed with the belief that some of them were probably not cases of actual paralysis, but of ankylosis.[3] This is especially likely to be true in such diseases as typhoid, erysipelas and other conditions attended with severe local inflammation adjacent to the joint, or. as in rheumatism and gout, with inflammation of the joint itself.

The possibility of error in deductions based upon the statistics of reported cases of paralysis, some of which may have been wrongly diagnosed, must therefore be taken into account.

The possible myopathic origin of loss of motion in the larynx must also be remembered. This condition may be present in wasting diseases, muscular atrophy, rheumatism and carcinoma.

While first asking attention to the better known departments of the subject, it is to the toxic conditions that I shall finally bespeak especial attention and interest, believing that the time has come when light should be thrown upon the obscurities which surround them and hoping that ere long more complete and definite knowledge may be secured.

The causes of recurrent laryngeal paralysis of peripheral origin as far as known may be divided into three general groups, namely:

1. Cases due to traumatism, or laceration of the nerve trunks.

2. Cases due to mechanical pressure upon the nerve.

3. Cases due to toxic influences, either of the nature of poisons ingested into the body, or of toxemias or infections generated by disease.

As has already been said, careful distinction must be made between actual paralysis and simple loss of motion due to myopathic causes. Also, from loss of motion from ankylosis of the crico-arytenoid articulations.

Of the various causes of paralysis of the recurrents, we will first consider traumatism.

Laceration of the nerve trunk may occur in any part of its course and from a variety of accidents and the injury may be inflicted from without the larynx and trachea or from within them.

The list includes incised, gun-shot and punctured wounds of the neck, self-inflicted or accidental; injuries to the nerve made in the course of surgical operations done upon neighboring parts; foreign bodies in the larynx, trachea or esophagus and unsuccessful efforts made at their extraction; injury to the nerve from attempts at strangling and from blows upon the larynx.

Division of the recurrent happens occasionally in cases of cut throat. The possibility of its existence should be recognized before attempting the complete closure of such wounds, as the effects of sudden urgent dyspnea might of course be very serious.

Lefferts[4] has reported a case of paralysis of the right recurrent nerve due to a punctured wound made by the point of the blade of a pair of scissors.

The recurrents are liable to injury in operations for the removal of various tumors, especially of the thyroid gland. This is particularly true of goitre, as a number of recorded instances will attest.

In view of the increasing popularity of the operative treatment of goitre this should be carefully remembered.

Operations for the removal of new growths of the neck in which the necessary dissections approach the vicinity of the recurrents may easily be attended with injury to them, as also may attempts at the extirpation of diseased lymph nodes lying near to the larynx or trachea.

The writer has seen cases of paralysis of the right side of the larynx following extensive extirpation of the cervical glands in recurrent carcinoma of the right breast.

The position usually occupied by the left recurrent behind the inferior thyroid artery renders it liable to injury during ligation of the latter.

We will now consider the subject of recurrent paralysis due to mechanical pressure.

The list of known causes includes: Disease of glands and of lymph nodes; new growths; aneurysms; diseases of the heart; diseases of the pleura and scoliosis.

Comparatively few cases of recurrent paralysis have been recorded in any country. At present reliable statistics as to the relative frequency of these causes are not to be obtained. Accuracy of statement therefore is impossible.

Clinical experience seems to prove that this division presents by far the largest number of the cases that come under observation. Scientifically speaking, they are more interesting than the cases due to traumatism, for while some are relatively common, others are rare; and while some—fortunately, as a rule, the more common ones—are generally rather easy of diagnosis, there are no small number which are very difficult, so obscure in fact that a positive diagnosis with the means of examination at present available to us seems to be impossible.

Many cases hitherto obscure have of late years been made clear by the aid of the Roentgen ray. Perhaps nowhere among the varied uses of that splendid contribution to science have more brilliant successes been obtained.

We will begin with the diseases of the glands and of the lymph nodes.

That a diseased condition of the thymus gland has ever been the cause of laryngeal paralysis I have not been able to learn.

Disease of the thyroid gland, however, is a common cause, occasionally from malignant involvement, but far more often from goitre.

Remembering the anatomic relations of the thyroid with the trachea it is easy to understand how the recurrents might be encroached upon by almost any swelling of that gland.

The size of the thyroid tumor seems to have little effect upon the degree to which it may endanger the recurrents. Thus a very large goitre may cause little or no dyspnea. On the other hand, one of small size may prove fatal.

The most striking instance of this which the writer has even seen came to his notice when he was Assistant Pathologist to the New York Hospital some years ago.

A middle-aged lady of rather full habit, while shopping, was suddenly seized with urgent dyspnea. She was hurried to the New York Hospital, which was nearby, and within a few minutes after the onset of the attack tracheotomy was performed. Before the canula could be inserted, she died.

At autopsy made by Dr. Geo. L. Peabody, the thyroid appeared to be slightly enlarged. Upon exposing it by dissection, a goitrous tumor, parenchymatous in nature, was found, the base of which did not measure quite four inches in diameter. It was so situated with regard to the trachea, however, that both recurrents had been pressed upon sufficiently to cause paralysis or, as is more probable, spasms of the larynx and asphyxia.

Disease of the tracheo-bronchial lymph nodes has been the cause of recurrent paralysis in a number of recorded cases. These may have become enlarged from syphilis, tuberculosis, malignant infiltration, or other infections.

In a case seen by the writer at St. Luke's Hospital, New York, a child, 2½ years old, suffered from urgent dyspnea, requiring a prompt tracheotomy, which failed to give complete relief. Death followed in a few days. The child was found to have general tuberculosis, and the cause of the dyspnea was a large lymph node pressing upon the middle section of the trachea. Great obscurity must often attend the recognition of these cases. The fact of their possibility, the presence of a condition capable of causing them, the absence of other discoverable cause and above all the use of the x-ray may all aid in arriving at the truth.

Malignant disease of the thyroid, the esophagus, the neck and the mediastinum is a common, if not the most frequent, cause of recurrent paralysis.

In all of these but the esophagus it is generally due to direct pressure upon the nerve trunk. While sometimes due to direct pressure from disease of the esophagus, it may also in the latter case be caused indirectly through the extension of the disease when located in the upper part of the esophagus to the larynx or trachea, so that both recurrents are surrounded by cancerous masses and thus totally paralyzed. Or the paralysis of the nerves may be direct from the pressure of degenerated cancerous glands upon them.

Sandby[5] has reported a case which has become famous for two reasons—first, because as a result of esophageal carcinoma adductor paralysis was first observed. Later this disappeared and a condition of purely abductor paralysis took its place. Finally the cords assumed the cadaveric position, thus proving the complete paralysis of the nerves. A second point of great interest in this oft quoted case is that it seems to contradict the so-called "law" of Semon as to the supposed vulnerability of the abductor filaments of the recurrent nerve.

In the minds of most practitioners, the discovery of fixation of the left side of the larynx is almost proof positive of the presence of an aortic aneurysm. Very often this view is correct. In the experience of the writer with the cases that have come under his observation, aneurysm of the aorta has been the most frequent cause of left recurrent paralysis. The mechanism of this is too familiar to require special explanation. It must be remembered that bilateral paralysis with its attendant dangers may be developed by the growth of the aneurysmal tumor in its late stages.

The position of the right recurrent with relation to the right subclavian artery is such as to render it vulnerable in case of aneurysm of that vessel. Such cases are very rare in comparison with those of left recurrent paralysis due to aortic tumor.

Aneurysm of the innominate has been said to be an occasional cause.

Cases have been reported in which recurrent paralysis has been caused by various diseased conditions of the heart, especially valvular disease in the form of mitral insufficiency and of pericarditis. By the use of the term "valvular disease" in this connection we are probably meant to understand the effects of it in the form of dilatation or hypertrophy, the increase in the size of the organ causing it to press upon the recurrents, as would be the case in pericarditis. Apropos of this subject, the case reported by Baumler[6] presents features of extraordinary interest.

A young man suffering from syphilis and an old mitral insufficiency lost his voice synchronously with the appearance of a pericardial exudation. Examination of the larynx showed a bilateral abductor paralysis. With beginning resorption of the exudate the voice gradually returned, at first rough and hoarse. Two weeks later it became entirely normal and remained so until his death.

From its position with regard to the apex of the right lung the right recurrent may be pressed upon under certain morbid conditions of that part.

Thus, in fibrous phthisis, such cases have been found. They have also been caused by thickening of the pleura covering the right lung apex. It is said that the same thing has been seen in the case of the left lung.

Finally, a few cases are reported in which paralysis was attributed to penumonia, pneumothorax and pleurisy. That the paralysis was actually due to pressure and not the result of severe peripheral inflammatory conditions, remains to be proved.

By far the most interesting group of cases which come within the scope of this article are those due to toxic influences. They are less frequently observed than those caused by traumatism or by pressure, so that opportunity to study them has not so often been given.

Moreover, the physiologic and pathologic processes by which they are brought about are far too little discussed and much less well understood. It is proper therefore that particular attention should be called to them both by reason of their pathologic importance and because they offer one of the most interesting and valuable fields for original research to be found within the range of medicine.

The ingestion of certain drugs and chemicals is found to produce in some cases paralysis of the recurrent laryngeal nerves.

In like manner recurrent paralysis occasionally accompanies certain acute infectious diseases. If it be true that the poison acts directly upon the nerve-elements through some especial affinity of the one for the other, then the variety of toxics capable of so acting is remarkable, and it suggests a vulnerability of the recurrents to toxic effects which is, to say the least, unusual and peculiar. Other sets of nerves may be similarly acted upon, as in poisoning from lead, alcohol or diphtheria. The number of different toxins which have been said to affect the recurrents, however, seems unusually extensive.

The list of causes of toxic paralysis of the recurrents should be divided into two groups: First, mineral and vegetable poisons. and secondly, toxins generated by certain acute infectious diseases. To the former belong lead, arsenic, antimony, copper, iodid of potassium, iodoform, and, it has been alleged, cyanid of potassium and phosphorus.

Recurrent paralysis due to other mineral poisons must be very rare. A few cases have been observed which seem fairly to have been due to the influence of arsenic, iodid of potassium and iodoform.

Those referred to antimony, copper, cyanid of potassium and phosphorus, are apparently of doubtful nature.

The first case on record in which the influence of lead upon the recurrents was observed by the aid of the laryngoscope was seen by Sir Morell Mackenzie[7] in 1865. He reported a case of complete paralysis of the right side of the larynx in a young man suffering from general lead poisoning. The paralysis disappeared after two months' application of electricity.

Our fellow member Sajous[8] in 1882 reported a case of bilateral paralysis of the larynx due to lead poisoning, and Seifert follows in 1884 with three well observed cases. A few others have appeared in literature up to the present time.

The cases reported in which other minerals have been thought to produce recurrent paralysis have been so few in number as to make deductions impossible. They suggest the possibility of coincidence quite as strongly as they do that of cause and effect.

Paul Heymann[9] collected over 50 cases of mineral poisoning of the larynx. He found lead to be the most common cause. Usually the abductors were affected.

Among the vegetable poisons, the following have been alluded to in the literature of the subject: alcohol, opium, morphin, belladonna, atropin, cannabis indica, cocain.

Among the vegetable poisons mentioned, alcohol properly heads the list. Hippocrates[10] calls attention to that form of aphonia of the drunkard which comes upon him in his cups and does not disappear when he has become sober. Several good authorities have quoted the coexistence of recurrent paralysis with alcoholism. Cases have also been reported as being due to opium and to belladonna, and it has at least been suggested that similar results may arise from cannabis indica and from cocain.

The questions raised by the action of the drugs and minerals just described upon that always peculiar pair of nerves, the recurrent laryngeals, are perplexing. In what manner do they inhibit the action of these most active parts?

1. Is there any truth in the supposition that there is an "elective affinity" existing in these particular nerves in favor of certain drugs through the influence of which the one exercises an inhibitory effect upon the other?

2. Does the drug produce changes in the nerve tissue itself which result in the loss of its activity?

3. Is the lesion a neuritis, induced by pressure from congestion of neighboring parts or otherwise?

5. Are the effects upon the recurrent due to central causes, the latter induced by the drug?

Turning now from the mineral and vegetable poisons which may affect the recurrents, we take up the consideration of the alleged results produced by the toxins of some of the acute infections:

These include typhoid fever, acute articular rheumatism, influenza, diphtheria; typhus fever, acute croupous pneumonia, puerperal fever, erysipelas, measles, scarlet fever, gonorrhea, serum therapy.

Of 73 cases collected by Sendziak, 26 occurred in typhoid fever, 18 in acute articular rheumatism, 11 in influenza, 9 in diphtheria, 3 in typhus fever, 2 in croupous pneumonia, and 1 each in puerperal fever, erysipelas, measles, scarlet fever, gonorrhea and the use of serum therapy.

Of the infectious diseases in which recurrent paralysis may be found typhoid fever seems decidedly the most common. The paralysis may be unilateral or bilateral. It may occur early in the course of the disease, but it generally appears towards its close, during the 3d or 4th week.

Osler,[11] agreeing with the generally received opinion, attributes it to a neuritis of the recurrent and states that the general activity of the toxins in a given case has more influence upon the nervous system in causing the neuritis than has the temperature. In other words, the neuritis is not so apt to be developed in a case where the temperature is running a high course as it is in one in which the typhoid intoxication is profound.

Laryngeal paralysis in a severe case of typhoid is, of course, an exceedingly grave complication, although in the milder cases of fever and where the paralysis is unilateral it is not so dangerous.

Regarding the cause of the paralysis, the question again arises as to whether, after all, the nerve irritation is not brought about by the violent surface inflammation, commonly met with in and around the larynx in this disease.

While most writers state that the paralysis of the recurrents in typhoid is generally due to neuritis, certain ones believe that the loss of motion is sometimes myopathic. Degeneration of the laryngeal muscles may take place in like manner with the degeneration of the abdominal muscles in typhoid.

Morris Schmidt[12] says that recurrent paralysis has occurred in typhoid patients suffering from complicating pleuro-pneumonia, glandular swelling and muscular degeneration.

May it not be possible that some of the instances reported as being due to typhoid toxemia have, after all, been caused by such complicating conditions?

In typhus fever, as in typhoid, recurrent paralysis may supervene. It is said to be more frequent in typhus than in typhoid; it is likely to occur at an earlier period of the disease and to be of a more serious nature.

In acute articular rheumatism recurrent paralysis has been found in all its phases: unilateral, bilateral, abductor and complete.

Here again the causes are variously stated, neuritis and rheumatic affection of the laryngeal muscles having the preference.

Clinton Wagner and others have reported cases of gouty inflammation of the crico-arytenoid articulation with perichondritis, necrosis, abscess and sloughing of the arytenoid cartilage. The possibility of such a case should suggest the possibility of rheumatic affection of the laryngeal muscles and admit of the assumption that not every case of loss of motion in the larynx is necessarily neuropathic.

Morris Schmidt[12] says that he has not infrequently seen recurrent paralysis following influenza. Other writers have reported a few such cases. This does not seem improbable in a disease capable of affecting so profoundly the nervous system in general.

Morris Schmidt[12] also calls attention to the fact that in diphtheria it is still questionable whether the resulting paralyses are due to central or to peripheral causes. In the opinion of the writer it is not necessary that fixation of the larynx following diphtheria should be due to paralysis at all, since under conditions of inflammation as violent as those often found in laryngeal diphtheria, ankylosis of the cricoarytenoid might easily be produced. In several cases seen by the writer of unilateral loss of motion present many years after an attack of diphtheria this has seemed to be the true explanation. The question is rendered still more difficult, however, when we remember that following a genuine paralysis the joint may become ankylosed through disuse.

Apparent paralysis of the larynx may also accompany the severe infections of the throat non-diphtheritic in type, as for example, the staphylococcus and the streptococcus. It seems more probable that the loss of motion is due rather to the effects of inflammation upon the muscles and joints of the larynx than to its effects upon the recurrents.

One case each is reported of recurrent paralysis from erysipelas, puerperal fever, scarlet fever, measles, serum therapy and gonorrhea. In all but the first the paralysis was unilateral.

The last named case has been elaborately described in an extensive and scholarly article by Lazarus.[13]

With regard to toxic paralysis, some very interesting questions arise.

1. If it be true that there is such a thing as a "selective affinity" between the recurrents and a given poison, in a case of poisoning we would expect to find both recurrents affected. In point of fact mineral poisons seem almost invariably to affect but one.

Again, if the toxins of infection cause paralysis by exciting a recurrent neuritis, we would expect the result to be bilateral, whereas a considerable proportion of such cases have been unilateral. Both of these facts seem to contradict the affinity doctrine.

2. Is it possible that toxins, whether inorganic or organic, produce special structural changes in the nerve tissue itself, which result in recurrent paralysis.

For the answer to this question we must look to the pathologist, although it would seem that simple inflammation of the nerve would be all that was necessary to inhibit its activity.

3. Are the effects of toxins upon the recurrent due to central causes? Here again the fact that bilateral paralysis is rare and unilateral the rule would throw doubt upon the probability of an affirmative answer.

4. Is the lesion a neuritis, induced by pressure from congestion of neighboring parts or otherwise? It seems probable that in many cases it is, since this would explain the presence of recurrent paralysis from so many different varieties of causes, most of which are associated with congestive conditions of the throat, and for its occurrence in so many different forms.

It is easy to suppose that an inflammatory condition severe enough to cause a crico-arytenoid ankylosis might cause a paralysis of nerves lying as near the surface as do the recurrents.

It is impossible to escape the conviction that some of the cases of recurrent paralysis found accompanying violent inflammations of the throat have been due to the effects of superficial irritation.

Nothing is more common than to find a paralysis of the posterior arytenoideus muscle accompanying an acute laryngitis. The position of this muscle near the surface and at a part of the larynx where the inflammatory conditions are apt to be most intense, would render its nerve filaments peculiarly liable to irritation and injury.

Percy Kidd[14] reported a case of bilateral paralysis of the larynx, cadaveric position, following a severe laryngitis, lasting about three weeks and ending in recovery.

Such cases are not unique. If they suggest anything, it is that surface irritation may be the cause of recurrent paralysis. In many of the diseases in which recurrent paralysis is reported to have occurred, inflammation of the throat is a marked symptom.

If it can be caused by the irritation of a simple laryngitis, it is certainly possible that the still more violent inflammation accompanying severe acute infections may produce it. Doubtless among both groups of toxic causes there are some which act in this way.

These are some of the questions that remain to be explained and finally answered. If my presentation of them has been imperfect and my deductions inadequate, it must be remembered that they have thus far baffled the efforts of all who have attempted their solution. With the advance of knowledge of the nervous system in general and with the increased accumulation of clinical material it is to be hoped that better progress may ere long be made.

A study of the literature of this subject shows that it leaves much to be desired. Considering the large variety of conditions which may cause it and the vast number of the cases which many of these conditions create, as for instance, traumatism, tumors, cardiac diseases, tuberculosis, typhoid, diphtheria, influenza, etc., it is strange that relatively so few recurrent paralyses have been recorded. Doubtless more careful observation, aided by laryngoscopic examination, would demonstrate many additional ones. Here, as is the case in many other departments, many of the histories have been too incompletely recorded to be of any value.

The latest writer, Sendziak,[2] claims to have found 1017 cases, 201 of which he says are his own. Where he found them he does not state. Unfortunately he does not give the references to them, so that his statistics cannot be verified, nor can the articles from which he derived his material be studied. His own articles being published in the Polish language and in obscure provincial journals, makes them almost impossible of access.

The article of Lermoyez is classic and well worthy of study. Paul Heymann's[9] monograph on recurrent paralysis, due to the effect of poisons, is excellent. It contains a full and very valuable bibliography.

BIBLIOGRAPHY.

1. Shurly. D. Appleton, 1900, p. 226.
2. Sendziak. Lwowski, tygod. lekars, 1907, Vol. ii, p. 17.
3. Delavan, D. Bryson. N. Y. Med. Record, Jan. 24, 1903.
4. Lefferts. Amer. Journal Modern Med. Sci., July, 1881.
5. Sandby. Brit. Med. Journ., March 12, 1904.
6. Baumler. Deutsch. Arch. für klin. Med., 1867, Vol. ii, p. 520.
7. Mackenzie, Morell. Hoarseness, Loss of Voice, London, 1868.
8. Sajous, Chas. E. Arch. Laryngology, Vol. iii, 1882, p. 58.
9. Heymann, P. Fränkel's Arch. für Lar. and Rhin., Band v, November, 1896.
10. Hippocrates. Opera Omnia, Editio Froesius, 1605, p. 352.
11. Osler. Nothnagel's System of Medicine.
12. Schmidt, Morris. 3d edition.
13. Lazarus. Fränkel's Arch. für Lar. und Rhin., Band v, November, 1896.
14. Kidd, Percy. Brit. Med. Journ., May 19, 1888.

XXIV.

THE ETIOLOGY OF OTOSCLEROSIS.*

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The secretary is to be congratulated on putting a subject as important as this on the program for discussion this year. It is my only regret that the duty of presenting the first paper in it should devolve on one so incompetent. Twenty years ago we heard and read little upon this potent cause of deafness. The textbooks of that day mentioned a form of middle ear disease known as dry or hyperplastic catarrh, but the impression was given that it was met with only infrequently and was of little significance compared with the common hypertrophic variety. Since that time otologists, especially those abroad, have busied themselves with the subject and an inspection of the literature of the past two decades will show many valuable contributions thereto. There is today in spite of all this, much unmerited ignorance and too often it is grouped clinically with the other non-suppurative affections of the middle ear.

Any intelligent consideration of the causes of otosclerosis demands a clear understanding of the exact pathologic process which is producing the affection. This since Politzer's valuable paper, later reinforced by Siebenmann's work, has commonly been held to be a rarefying process of the labyrinthine capsule or the region in the vicinity of the oval window either internal or external to it or both. This is regarded by Politzer to be a primary affection. That such a condition at times exists cannot be gainsayed as has been abundantly shown on the postmortem table. That it is the usual condition in the cases which we are accustomed to regard clinically as otosclerosis, is. however, decidedly open to question. The grounds for this radical departure from what has been a generally accepted position of all writers, are clearly set forth in Lucae's recent treatise on the subject entitled "Die Chronische Progressive Schwerhörigkeit."

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*Read at the meeting of the American Otological Society at Atlantic City, June 24, 1908.

Lucae calls attention to the fact that von Troeltsch was the first to make use of the term sclerosis, and while the latter does not specifically mention it, he in all probability based his employment of the term on the careful description of a group of cases by Toynbee which he characterized as cases of rigidity of the lining membrane of the ear. Adopting Toynbee's idea of the pathology in such cases and defending his position at much length and by the citation of numerous cases of his own, Lucae advances the theory that a previous inflammatory condition, either the ordinary hypertrophic form or that of suppuration, is at the bottom of the great majority of all cases of sclerosis. Such a condition, especially one of transient suppuration occurring in childhood or infancy, as it often does, can most easily be lost sight of at the period when the symptoms of the disease are first noted. Toynbee's description of these so-called cases of "rigidity" of the mucous membrane of the ear is deserving of being quoted in full: "The mucous membrane of the tympanum may be subject to chronic inflammation, complicated or not with rheumatism, at any period of life, and, if neglected, this is liable to terminate in a rigid state of the membrane; so that the ossicles become bound together more firmly than is natural. In advanced years, this rigid condition seems to take place without any symptom of inflammation. The membrana tympani also partakes of this rigid condition, and, what is of far greater importance, the base of the stapes may become much more firmly fixed to the border of the fenestra ovalis than is natural. As a consequence, the membrane of the fenestra rotunda and the fluid of the labyrinth participate in this fixed condition. This affection is less prevalent in the young than in the adult, in whom it is liable to occur after repeated attacks of cold, whether with pain or not. It is, however, most frequent in persons advancing in life, and may, in fact, be considered as the disease which causes deafness in advancing years. The generally received opinion that in this kind of deafness the nervous system is at fault, is manifestly incorrect, as proved by the symptoms and by the mode of relief found beneficial." In addition to this condition so clearly set forth, Toynbee describes two others giving rise to similar symptoms. First, membranous bands in the tympanic cavity. "These may arise in any part of the cavity connecting together the ossicles and attaching them to the walls of the tympanum and to the drum membrane." The stapes is wont to be bound down to the promontory and thus a species of ankylosis produced. Toynbee is of the opinion that these bands are the result of the effusion of fibrin from the membrane or from the adhesion together of the already hypertrophied membrane.

The second condition is that of ankylosis of the stapes, so well known to all aurists since his day. In his opinion, however, this ankylosis permits of various gradations according to the stage of the disease. At first it is only partial without expansion of the base of the stapes, depending on a rigidity of the capsular ligaments. This is the earliest form. A second stage is the expansion of the articular surfaces without changes in the bone. This was found in 49 of the 136 specimens of ankylosis described in his catalogue. The later stages represent hypertrophy of the entire stapes base. Here it is often impossible to remove the crura from the oval window without breaking, and finally we may meet in the most advanced cases, actual deposit of bone.

These classical views of one of the earliest workers in our specialty, we have taken the liberty of quoting at some length because of their direct bearing on the question of etiology under consideration and because they serve without doubt as a basis for the views which have been set forth by Lucae in his recent book just referred to. Lucae is prepared to admit that a certain percentage of ear cases will fall in the group of primary disease of the labyrinthine capsule but believes that careful inquiry as to a previous ear condition and the employment of the more accurate methods of diagnosis, will serve greatly to reduce the number. The opinion of so competent and experienced an observer, is worthy of our most thoughtful consideration. While not prepared perhaps to subscribe to it at present in its entirety, we feel that it represents in the main a reasonable view of the subject, one that offers hope as far as prognosis is concerned where hope formerly could not be entertained and one entitled to be applied to all cases of this class which present themselves to us for treatment.

We would repeat then that in our opinion otosclerosis represents not one but several pathologic conditions. Viewed from this standpoint the question of cause or causes becomes a broad one. Inasmuch as there seems in the majority of cases to be a precedent middle ear catarrh, it may be said that the same causes will tend to produce both. It is generally agreed that the most common cause for the former is disease of the oro- and epi-pharynx, and that the extension to the ear is wont to take place through the Eustachian tube. To this view we have not the least wish to take any exception. Indeed, experience only impresses upon us the close relation which exists between the two localities, but as to the equally popular teaching of the importance to be attached to affections of the nose in their effect on the ear, we have still to be convinced. At the same time we are willing to admit that marked obstructions in the nares can be accountable for a certain number of ear cases. Their number in our judgment is, however, small in comparison with the number due to other cases. Moreover it must be constantly borne in mind that we continually meet patients with complete nasal obstruction due to polyps or other neoplasms without any aural symptoms whatever. Indeed, we would go further and say that we believe that in many instances where is well defined throat lesion, the ear disease is not dependent upon this but that there is a common cause producing both.

In line with what has just been said regarding the relation of otitis media chronica hypertrophica to sclerosis and bearing on the question of a common cause for affections of the throat and ear, we may be permitted to refer to the statistics of 21 cases of undoubted sclerosis seen recently in private practice. Many of them had undergone operations upon the nose or throat without benefit before coming to us, and what is of particular interest, in not one of them at the time of our examination was there what might be described as a gross nasal lesion, while in most there was no complaint addressed to the nose at all. Yet in 15, or over 66 per cent, there was a clear history of repeated colds extending over several years. This complaint of repeated colds is the most significant feature of the history of many of these cases and one upon which the greatest emphasis is to be placed. The cause in our judgment in the majority of instances is not to be found alone or chiefly in the nose, as is shown by the failure to relieve by nasal treatment in the cases just quoted and in many others with which you all are acquainted. This cause without question varies in the particular case. Rheumatism and gout have always been regarded as among the more common contributing causes. Our experience with gout in this country is of necessity limited and we do not recall finding many cases where a well-established rheumatic history was obtainable. Rather do we think that emphasis should be put upon that condition which is often in the lay mind confused with rheumatism and which very possibly is at the bottom of many cases, i. e., lithemia or collemia as described by Haig, that condition of the system where a state of autointoxication exists due to the retention of certain products of excretion in the blood. We know that this accounts in no small degree for many cases of throat difficulty and there is no question in our own mind that a similar cause operates not infrequently to set up or aggravate trouble in the ear. Another widely acting agent in exciting both pharyngeal and aural inflammation and one that we have yet to grasp the full significance of, are bacteria. Most writers who have looked into the subject, are beginning to believe that there is no difference etiologically between an acute catarrhal otitis and an acute suppurative otitis except in the virulence of the bacterial invasion. In addition to rheumatism, many other constitutional diseases have been suggested as exciting causes. Among these may be mentioned tuberculosis, syphilis, struma, arteriosclerosis, and trophoneurotic changes which occur in tabes and allied affections. All can and do undoubtedly exist in connection with otosclerosis but the direct causal relation is yet to be shown except as any profound constitutional disturbance must affect every organ of the body and render it more liable to disease. The female sex is especially prone to the disease, which would seem to suggest the probability that a highly organized nervous system, such as is wont to be found in women, is an important predisposing factor.

Age would appear to have some bearing upon the onset of the disease. At least it makes its appearance in the majority of cases in early manhood and womanhood. In 733 cases reported by Lucae, it developed in 74 per cent between the years of 21 and 50. In 87 per cent of these, the affection was double sided pointing to the general character of the malady.

Pregnancy has been advanced as a possible cause. While it does undoubtedly occur that marked flagging of the hearing occasionally is noted at that time, it can only be regarded as an instance of the now well recognized intimate relation between the sexual and sensory systems.

More striking than all other causes save the one of throat disease, is heredity. There can be no question that heredity plays an important role in otosclerosis. In 641 cases where records on this point were kept by Lucae, 235 or 37 per cent gave such a history, while Bezold puts it as high as 52 per cent in his cases. When it is considered how difficult it is to obtain accurate information regarding this due to the early death or separation of the parents or grandparents, there can be little question that the figures of Lucae are too low rather than too high. Highly significant as bearing on the question of the frequency of a preceding chronic catarrhal otitis in the majority of cases, is the fact that of the 235 cases where heredity was shown, 77 per cent had demonstrable pathologic changes in the tympanic cavity. This would seem to prove conclusively that inherited otosclerosis does not differ at all in this particular from the non-inherited variety. Quite in line with this observation and serving at least as a most ingenious explanation for this widely recognized peculiarity, is the theory of von Troeltsch that the family likeness is to be found as far as the ear is concerned in a uniform narrowing of all its measurements predisposing as soon as any inflammatory process has appeared, to an early throwing out of adhesive bands and the binding down of the ossicles in consequence. Siebenmann has also advanced the view that there exists an abnormal post-embryonic developmental process. Others have tried to show that the disease is especially wont to develop in those who are anemic or disposed to worry. Certain it is that some general underlying predisposing cause is needed to explain so widespread and characteristic a feature of the disease.

A careful and painstaking review of most of the recent articles on the subject, leads us to feel that not enough attention has been paid to this phase of the question and in order that more definite and accurate data may be secured, we would urge that in all our future cases we endeavor to secure the fullest information possible on every point which may however remotely assist in arriving at an authoritative answer to the all important question in the consideration of any disease, namely, what is the cause?

XXV.

PATHOLOGY AND PROGNOSIS OF CHRONIC PROGRESSIVE HARDNESS OF HEARING—OTOSCLEROSIS.

By W. Sohier Bryant, A. M., M. D.,

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This title assigned to me is somewhat misleading; I should prefer to substitute "Pathology and Prognosis of Chronic Interstitial Otitis." I am glad to have the opportunity to express my views on this subject, since I think confusion has arisen by the loose way in which the terms otosclerosis and chronic progressive hardness of hearing have been used by various authors. I use otosclerosis to denote an interstitial inflammatory sclerotic condition of the ear especially characterized by vascular changes and usually located in the tympanic cavity or in its walls. The term otosclerosis is in no way expressive of the vascular ostitic lesions to which it is often applied. On the other hand, I use the term chronic progressive hardness of hearing to denote a class of ear affections due to changes in the middle ear or its walls, these changes being independent of ulcerative processes but characterized by chronicity and a tendency to increasing deafness. In exceptional cases the hearing appears to remain stationary for long periods. Most of the cases, all except the most advanced, may show arrest of the process and a degree of improvement under proper therapeutic and surgical management. Some of the synonyms used for chronic interstitial otitis are: (1) dry chronic middle ear catarrh (Schenke); (2) otosclerosis; (3) chronic progressive deafness; (4) otitis media insidiosa; (5) rarefying ostitis of the labyrinth capsule. None of these terms is good. "Dry middle ear catarrh" does not express the pathologic condition. "Otosclerosis" is an expressive term but it has been ruined by its erroneously restricted application. "Chronic progressive deafness" has also been misapplied. "Otitis media insidiosa" is a rare form of chronic interstitial otitis and the term is not sufficiently comprehensive. "Rarefying ostitis of the labyrinth capsule" is inadequate since it defines only a local terminal condition of the general pathologic process. My only excuse for mentioning these different terms is an attempt to explain away a misunderstanding in order to go back to the fundamental etymopathologic meaning of the words. For the general pathologic condition we are considering I would prefer to use the term chronic interstitial otitis with a special adjective referring to the parts affected in any one particular case, as otitis interstitialis chronica membranae, otitis interstitialis chronica ossiculae, otitis interstitialis chronica promontorii et fenestrae ovalis, otitis interstitialis chronica labyrinthi.

Coming back now to our subject, the results of clinical observation and treatment show that chronic interstitial otitis is due to middle ear, tubal and nasopharyngeal disturbances in many cases and to constitutional causes in others, or to all combined. The nasopharyngeal disturbances seem to work in a reflex way through the complicated nerve connections of the tympanum. Ever since I took up our specialty I have paid a great deal of attention to this subject and the overwhelming evidence of my clinical observation and experience together with laboratory work have forced me to accept the following views:

I. PATHOLOGY.

The pathology of the condition under discussion is dependent upon vascular disturbances affecting the blood supply of the tympanic cavity and walls, with resultant trophic changes. These pathologic processes have been described by many authors. The alteration in the blood supply causes either a hyperemia or anemia. Either of these conditions may be primary, but the anemia alone can be secondary. The primary anemia is of vasomotor origin and is usually found in young females. It has been described under the name of "otitis media insidiosa," a rare condition. The disturbance of the blood supply may be general or confined to one or more of three regions: (1) The drum membrane; (2) the major ossicles; (3) the stapes and labyrinthine wall. The changes are in most cases unequally distributed throughout the tympanum and in some cases may be sharply defined and limited to very small areas. Different stages are usually present at the same time. For instance extreme atrophic thinning, degeneration, calcification and fibrous thickening may be present in the same membrane. These circumscribed areas of different processes give a mottled appearance to the tympanic membrane.

There is nothing peculiar to the trophic changes caused by the altered blood supply of the middle ear. These changes are identical with the results of similar causes in other parts of the body, namely, congestion, vascular proliferation, infiltration, interstitial inflammation, hyperplasia, contraction, compression of blood vessels, anemia, degeneration, calcification, vascular ostitis or osteoporosis and hyperostosis. The peculiar feature exhibited by the middle ear is the very limited and sharply circumscribed area of the changes. The reason why similarly circumscribed changes are not found in other regions is that nowhere else do similar anatomic relations occur. The almost terminal arterial supply of the tympanum and its walls, where the mucoperiosteum of the tympanum admits of a blood supply running in one plane only, tends to render the blood supply subject to extreme variation over small areas. This tendency is enhanced by the peculiarly complicated arrangement of the tympanic plexus of sensory, motor and sympathetic nerves. Similar anatomic arrangements are not found anywhere else in the body. The tympanum has another peculiarity; wholly independent of central influences, it can control its own blood supply through its ganglionic connections. This control is brought about by the direct connection of the carotid and the minor vascular plexuses of the tympanic system through the intervention of the sensory nerves of the drum cavity. The nerve ganglia connected with the ear make it more subject to vasomotor changes due to local causes, than any other regions can possibly be. Among the most important causes of hyperemia are infectious processes of all grades whose resultant cicatricial contractions produce local anemia.

Because the mucous membrane is the most liable to vascular change, the pathologic changes under consideration affect this first; next they affect the submucous or periosteal layer, and last the bone. The pathology is exceedingly simple, but owing to the variety of structures involved and the location of the processes the resultant subjective and objective effects are quite varied. Certain areas of the tympanum, the oval window, promontory and drum membrane are most often affected because these areas are more subject to reflex vasomotor and other vascular changes. These parts also are more apt to attract attention because of marked symptoms.

The congestive stage of the process is accompanied by vascular increase and later cellular infiltration; increase of fibrous tissue results, the contraction of this new connective tissue shuts off the blood supply and nutrition suffers with signs of degeneration and loss of pliability of the connective tissue. After the congestive stage is passed the mucosa, mucoperiosteum and drum membrane have thickened and become grayish white. Later there may be a deposition of lime salts in the fibrous and periosteal layers. Osteoporosis follows the vascular ostitis or congestive stage in the bone. The diminished blood supply of the periosteum and bone caused by fibrous contraction and shutting of the vessels results finally in absorption of bone or osteoporosis and in the end hyperostosis occurs following the bone absorption and in conjunction with it.

Gross examination and histologic findings of the tympanum show one or more of the above mentioned pathologic conditions which take the following forms: (1) The congestion of the drum membrane due to an engorgement or an increase of the vessels of the mucosa, later similar changes of the membrana propria; (2) anemia of the drum membrane due to contraction or compression of the vessels; (3) opacity of the drum membrane due to a loss of transparency of the mucous or fibrous layers from cellular infiltration, fibrous thickening or degenerative processes and calcification of the connective tissue; (4) thickening of the drum membrane accompanied by opacity and due to the cellular infiltration and an increase of the connective tissue element; (5) thinning of the drum membrane due to atrophy of the mucous layer and of the membrana propria; (6) functional impairment of ossicular sound transmission from congestion and edema of the ossicular articulations, especially of the orbicular articulation of the stapes; (7) contraction of fibrous adhesions of ossicles, drum membrane or tympanic walls. These adhesions are due to hypertrophy or contraction of the normal mucous membrane, reduplications or to cicatricial adhesions; (8) rigidity of the articulations of the major ossicles and stapes due to atrophy and degeneration of fibrous tissue; (9) congestion and cellular infiltration of the promontory similar to the corresponding changes in the drum membrane; (10) functional rigidity or impairment of motion of the stapes without ankylosis due to congestions and cellular infiltration; (11) rigidity of the stapedio-vestibular articulation and fibrous ankylosis due to contraction of new fibrous tissue; (12) bony ankylosis of the foot plate of the stapes due to hyperostosis of the stapes and oval window; (13) vascular ostitis and osteoporosis of the ossicles and tympanic walls; (14) hyperostosis of the ossicles and tympanic walls; (15) hyperostosis of the labyrinthine walls, encroaching upon the labyrinthine spaces and interfering with the functions of the membranous labyrinth.

The bone changes begin with a thickening of the mucoperiosteum due to increased vascular formation and cellular infiltration. The nutrient vessels of the bone undergo similar changes. Later the new connective tissue formed from the infiltrating cells contracts, compresses the vessels and lessens the blood supply and interferes with the nutrition of the bone causing the formation of lacunae. Slightly later in the process but concomitant with the vascular ostitis the periosteum commences to form new bone in the form of irregular hyperostoses. The bone changes are most apt to commence in the neighborhood of the oval window and are usually found in the outer labyrinthine wall.

The pathology is founded on the etiologic basis of primary and secondary disturbed circulation, due to local or general causes. Because of the notoriety certain authors have brought upon these conditions and on account of the nomenclature erroneously applied to them, the pathology of the bone changes taking place about the stapes and in the labyrinthine capsule and other parts of the tympanic walls and temporal bone deserves special mention. Many authors have been surprisingly unanimous in their observation concerning the pathology of the condition rightly called rarefying ostitis. When, however, it comes to the steps which lead to this final condition, there is a great variance of opinion which increases when the primary pathologico-etiologic factor is under consideration

Many authors maintain with plausible arguments that this condition is a true bone syphilis; others equally plausibly disprove the presence in many cases of syphilitic infection. What then is the explanation? If we compare the progressive pathologic steps and the histologic findings with the course and result of syphilitic bone inflammation, we find an absolute identity of changes and histologic pictures. First there is an engorgement of the blood vessels followed by interstitial infiltration of round cells causing an increase of connective tissue elements. Then contraction of the connective tissue takes place shutting off the blood supply, next vascularization and vacuolization of the bone commences and finally new bone is formed.*

We know that the tympanum is subject to vasomotor changes which finally cause diminution of blood supply and atrophic changes. We know from frequent observation that the oval window and promontory are especially liable to chronic congestive states. Why should not the congestion produce the same result on the promontory as elsewhere, and why should not this result take the same form and produce the same alterations as similar vascular changes do in other bones? In default of a more logical primary pathologic cause for vascular ostitis of the labyrinth capsule, I believe it may perhaps be due to syphilis in some cases, but in the great majority of cases I believe it is due to altered blood supply of the lining mucoperiosteum of the inner wall of the tympanum independent of syphilis. Chronic interstitial otitis, including cases previously described under otosclerosis and spongifying ostitis of the labyrinth capsule, may follow, as we have seen, suppuration and infection as well as vasomotor changes and so-called "chronic middle ear catarrh," and therefore chronic interstitial otitis is a comparatively common affection. The pathologic changes in chronic interstitial otitis following suppuration are due to the diminished blood supply caused by interstitial infiltration brought on by the suppurative inflammation which ends in fibrous contraction. That rarefying ostitis should be hereditary is natural, since it depends on nervous instability and morphologic irregularities, qualities which are hereditary to a degree.

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*Schenke. Anatomischer Beitrag zur Frage der bei dem trockenen chronischen Mittelohrkatarrh (Sklerose?) vorkommenden Knochenerkrankung des Schläfenbeins (chronische vaskuläre Ostitis Volkmann), etc. Arch. f. Ohrenheilk., Vol. 53, p. 68, and plates, 1901.

Haberman. Zur Pathologie der sogenannten Otosklerose. Archiv f. Ohrenheilk., Vol. 60, p. 37, and plates, 1903.

Parrot. La Syphilis Héréditaire. Masson, Paris, 1886, plate 20, fig. 1.

II. PROGNOSIS.

The prognosis of chronic interstitial otitis is foreshadowed in the pathology. Without treatment the prognosis indicates a more or less gradual progressive deterioration of hearing. Under treatment the prognosis is favorable for improvement or prevention of further loss of hearing unless the pathologic changes are already far advanced. This improvement is possible because the fundamental causes of the altered blood supply which is the primary cause of the interstitial otitis are either local, nasopharyngeal or general conditions amenable to special treatment. If the blood supply is rectified before tissue changes have taken place, no sign will be left of the disturbances. The prognosis for the revitalization of degenerated tissues is dependent upon the prognosis for the improvement in the blood supply and our power to regulate the local vasomotor functions. If vasomotor regulation is established and the blood supply is corrected after tissue changes have taken place, the prognosis depends upon the ability of the patient to respond to the improved condition and to overcome the existing pathologic changes. The improvement in the ear following the improvement of the blood supply may extend to a complete restoration of function and return of the normal appearances of the drum membrane and ossicular chain. The prognosis is good if the congestive stage has not been passed, and good also after the progressive tissue changes have commenced up to the time the degenerative changes are very far advanced. The prognosis for the improvement and correction of adhesions by cutting and stretching is good. The prognosis for overcoming ankylosis is good if bony ankylosis and extreme degenerative states have not taken place. In bony ankylosis the prognosis is absolutely bad.

These previously published cases are illustrative of the prognosis in the more doubtful or advanced conditions:

1. Case No. 14229. A man, aged 67. Deafness commenced eight years ago; hearing has been very bad for three years; this condition was possibly aggravated by business failure. Hearing variable. Drum membrane whitish and opaque. Position, contour and light reflex normal; malleus movable. Politzer's acoumeter heard in the right ear at 4 inches, in left ear at 3 inches. Loud conversation heard in right ear at 6 inches, in left ear at 32 inches. Watch not heard in either ear. Bone conduction much shortened. Low limit for tone perception by bone, 128 single vibrations. Low limit for tone perception by air conduction above 256 single vibrations which were not heard by air. High notes well heard. Treatment by general hygiene and regulation of blood supply of the middle ear. Six months later, Politzer's acoumeter heard in right ear at 7 feet; watch at 20 inches; in left ear, Politzer's acoumeter heard at 4½ feet. Improvement has continued now for a period of four years.

2. Case No. 13083. A man, aged 47, had noted deficiency in hearing for eighteen years. Drum heads good color, fair contour and position, light reflexes very small. Nares partially occluded by hypertrophies and irregularities. Fossae of Rosenmüller partially closed by adhesions. Tubal mouths slightly obstructed by thickened mucosa. Watch heard in right ear at 15 inches; in left ear at 4 inches. Astringents and irritants applied to nasopharynx. Three years later watch heard at 48 inches in left ear and 84 inches in right ear. Improvement maintained.

3. Case No. 13036. A man, 24 years old. Family history of deafness. The attack commenced one year ago. Drum membrane normal. Buzzing tinnitus, and sounds like the ocean and various other noises. Politzer's acoumeter heard at 12 inches. Treatment: Astringent applications to the nasopharynx. Fifty-two days later, Politzer's acoumeter heard at 8 feet. Tinnitus very faint. The improvement has continued.

4. Case No. 13071. A woman, 74 years old. Hearing has been diminished some years. Drum membrane has small reflex and good color. It is contracted but not retracted. Congestion of promontory. Politzer's acoumeter heard in right ear at 10 feet; in left ear at 27 inches. Watch not heard. Intranasal and nasopharyngeal treatment for

thickened mucosa with stimulating astringents. Twelve days later, Politzer's acoumeter heard in right ear at 15 feet; in left ear at 42 feet. Two years later same watch heard at 4 inches in both ears. Improvement well held; very slight decrease during four years due to senile change.

CONCLUSION.

Pathology.—Exclusive of infectious conditions, there is but one fundamental pathologic condition in the middle ear which frequently interferes with normal hearing, namely, chronic interstitial otitis. All the various forms of atrophic processes, "chronic dry catarrhs" and "ostitis spongiosa of the labyrinth" come under this head. The nutritive changes may be inaugurated by local, reflex or general causes. The local causes include inflammation, infection and suppuration. The general causes work through nervous and blood supply changes.

The prognosis indicates a more or less gradual deterioration of hearing. Under rational treatment the prognosis is good for arrest of the progressive deterioration of hearing and improvement of audition in inverse ratio to the advances already made by the pathologic changes.

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XXVI.

SYMPTOMS AND DIAGNOSIS OF OTOSCLEROSIS.

By Geo. E. Shambaugh, M. D.,

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An insidious onset of deafness, beginning usually in early middle life, involving often but one ear primarily, yet as a rule including later both ears, associated usually with tinnitus aurium, and leading frequently after the lapse of years to profound deafness, this forms the usual clinical picture of the condition known as otosclerosis.

The gradual onset forms one of the most characteristic symptoms and on account of this insidious character it frequently happens that when the process is limited to one ear, and especially where tinnitus aurium is absent, the deafness becomes far advanced before the patient is aware that he has any defect in hearing. On the other hand in exceptional cases the progress of the defect in hearing is quite rapid, so that in the course of a few months' time the defect already becomes very marked.

There is no other form of ear trouble which so frequently involves both ears as does otosclerosis. This conforms with our knowledge of the etiology that some underlying hereditary condition is the all important factor. Still it is not uncommon for the process to be confined to one ear for a number of years, producing a profound defect in the hearing in that ear before anything can be detected on the opposite side.

Tinnitus aurium in some form is usually present. Its onset may antedate any detectable disturbance in hearing. On the other hand it may appear after the defect in hearing is well advanced. It may be intermittent, although as a rule it is constant. In pitch it may be high or low, and it is not uncommon for patients to complain of several quite distinct types of tinnitus at the same time. I have several times seen patients who described three distinct types of subjective noises with sometimes the one and at other times another temporarily increasing, so as to practically drown out the other two. A temporary increase in the subjective noises is usually associated with an increase in the deafness.

The deafness does not by any means invariably become severe. Often the progress of the condition is either so slow that no increase in the deafness can be detected for a number of years, or else there are periods of longer or shorter duration when the condition remains practically stationary.

There are other symptoms which are complained of with more or less frequency by patients suffering from otosclerosis. These are a sensation of fullness in the ear, slight pain deep in the ear, alteration in the severity of the deafness and the subjective noises depending on the condition of the weather or the presence of physical or mental fatigue. These patients are also frequently subject to slight attacks of vertigo attributed to an involvement of that part of the labyrinthine capsule covering the semicircular canals and the vestibule. Paracousis Willisi is more marked in otosclerosis than in any other form of ear trouble. Objectively the occurrence of Schwartze's symptom is the most significant. This consists in the presence of a reddish glow transmitted to an unusually transparent membrana tympani from a congestion of the mucous membrane covering the promontory. This reddish glow may be constantly present or it may be brought out only after catheterizing the Eustachian tube.

In the diagnosis of otosclerosis this condition must be distinguished on the one hand from cases of nerve deafness, and on the other from deafness due to middle ear disease.

In the diagnosis from nerve deafness it will be convenient to consider separately three distinct types of otosclerosis. First, those cases where the defect in hearing is due entirely to the fixation of the foot-plate of the stapes; second, those cases where in addition to the defect in hearing caused by the fixation of the foot-plate of the stapes there is also a more or less extensive defect in hearing caused by an involvement of the structures in the cochlea, and third, those cases of otosclerosis where the spongifying of the capsule of the labyrinth has produced no interference with the action of the foot-plate of the stapes, and where the defect in hearing is therefore due solely to disturbance in the function of the structures in the cochlea.

In the first group of cases, where the defect in hearing is due solely to the fixation of the foot-plate of the stapes, the diagnosis from nerve deafness presents no difficulties for even in the incipient stages of this process, and where the defect in hearing for the voice is but slight the interference with the action of the stapes already produces the prolongation of the bone conduction, the shortened positive, or even negative Rinne, and the elevation of the threshold for the perception of the lower tones which are characteristic of an obstruction in the conducting mechanism. In nerve deafness just the opposite reactions are found. Here the duration of bone conduction is more or less shortened, provided both ears are involved. The Rinne is always positive, when the tuning fork can be heard at all, except of course in those cases of nerve deafness which are limited to one ear where the Rinne will be negative, provided the defect in hearing is at all marked. In partial nerve deafness, too, the defect in hearing is limited, as a rule, to the upper part of the scale, although of course it is not impossible in nerve deafness to have a disturbance limited to the upper coils of the cochlea with a defect for the lower tones while the higher tones will still be heard normally.

In the second group of otosclerotic cases, where in addition to the defect in hearing produced by fixation of the foot-plate of the stapes, there is also a more or less extensive defect due to involvement of the structures in the cochlea, the diagnosis is often by no means so clear. This is particularly true in those advanced cases where the labyrinthine deafness is extensive, a condition to which the term disacousis has been applied.

The underlying pathologic process in otosclerosis, the spongifying of the bony capsule of the labyrinth, while beginning in most of the cases in the immediate neighborhood of the fenestra vestibuli and in this way producing usually an early fixation of the foot-plate of the stapes, is not always restricted to this part of the labyrinth capsule. The same change may take place in other parts of the capsule as well, producing as we have already pointed out the disturbance in equilibrium, when the vestibular part of the capsule is involved. In the same way, apparently, disturbances in the function of the organ of Corti are produced when the adjacent capsule of the cochlea becomes involved in this spongifying process. These disturbances in the cochlea appear to be circumscribed, and limited in a measure at least to the areas in the cochlea in more or less close relation to the part of the capsule involved. It is probably in this way we have produced in most of the typical cases of otosclerosis, even in the incipient stages, a defect for the higher notes of the Galton whistle, because that part of the organ of Corti stimulated by the higher notes of the Galton whistle lies under the promontory in close relation to the fenestra vestibuli, in other words, in close relation to the part of the labyrinth capsule involved in the spongifying process. Bezold has found cases of otosclerosis with circumscribed defects in the middle of the scale for part of the Galton whistle, and I have this past year studied a case of probable otosclerosis in which was found a circumscribed tone island as well as a circumscribed defect in the midst of the tone scale.

With the knowledge of the fact that in otosclerosis the disease of the capsule of the labyrinth is not always limited to the region of the fenestra vestibuli, but may spread more or less extensively throughout the capsule of the cochlea, producing a loss of function of the part involved, we can readily understand these cases of disacousis where in addition to the defect in hearing produced by the fixation of the foot-plate of the stapes there exists a more or less extensive involvement of the capsule of the cochlea with symptoms of nerve deafness. These cases fall naturally into two groups: first, those cases where in addition to the defect in hearing produced by the fixation of the stapes there is a defect due to a more or less extensive involvement of the basal coil of the cochlea. In these cases we will find in addition to the elevation of the lower tone limit a more or less extensive defect for the higher notes of the Galton whistle, yet the Rinne will be found to be negative and there will be found a prolongation of the bone conduction.

The left ear in the following case is an example of this group. Mr. B., 54 years old. His father's mother was deaf for many years. His father had a sister 70 years of age, who was quite deaf. Of her six children two are somewhat hard of hearing. The patient's father began to be troubled with impaired hearing at the age of 25. He lived to be 73 years old, and suffered for many years from severe deafness. The patient has one sister 44 years of age, who has been hard of hearing for fifteen years. He has one brother 50 years old, whom I have examined, who is suffering from an advanced stage of otosclerosis with more or less extensive nerve deafness, as evidenced by a marked defect in the upper part of the scale. The patient himself began to notice loss of hearing when he was 14 years old, first in the right ear, but later in both. He has always had some tinnitus aurium, much worse in previous years than lately, and Paracusis Willisi is very strongly developed. An examination found the drum membrane practically normal with slight thickening, and the Eustachian tubes normal. Whisper could not be heard in either ear. Spoken voice "28" heard somewhat clearer in left ear. In the Weber test the A fork is lateralized indistinctly to the left ear. The Schwabach test shows marked prolongation of bone conduction. Rinne for the a^1 fork is strongly negative for both ears. The lower tone limit for right ear is 30 d.v. There appears to be in this ear an indefinitely limited defect in the scale between h and c^4, and a well marked tone island from c^4 up to mark "14" on the Edelmann-Galton whistle. From "14" to upper limit there is a total defect. In the left ear the lower tone limit is 40 d.v. All tones above 40 are heard up to mark "11" on the Edelmann-Galton whistle. Above "11" there exists a total defect. In this case the defect in the lower part of the scale, that caused by the ankylosis of the stapes, is greater in the left than in the right ear, but the defect throughout the scale and at the upper limit, that caused by the involvement of the cochlea, is more extensive in the right ear. The result is that the patient relies on the left ear exclusively for hearing, and it is to this ear that the bone conduction is lateralized in the Weber test.

The second group of cases of disacousis includes those where in addition to the defect in hearing produced by the fixation of the foot-plate of the stapes and the loss of hearing for the higher tones produced by the spongifying of the promontory there exists extensive nerve deafness throughout the middle of the scale, evidently because of the involvement of the capsule of the cochlea at a distance from the oval window. The right ear in the case just cited is an illustration of this group. The functional tests in these cases, provided the hearing in the opposite ear is gone or both ears are similiarly involved, may give all the typical reactions due to nerve deafness, that is a pronounced defect in the upper part of the scale for the Galton whistle, a shortened bone conduction, and a positive Rinne for the a^1 fork and in addition there will be the marked elevation of the lower tone limit due to the fixation of the stapes. Whether the Rinne will be positive or negative in such cases depends upon whether the deafness for the particular fork used in making the test is part of the defect in hearing resulting from the fixation of the stapes, or whether the deafness for this fork is the result of disturbance in the cochlea due to the spongifying of its bony capsule. In the first case the Rinne will be negative while in the latter it will be positive. This also applies to the results obtained in making the Schwabach test. The duration of bone conduction may be prolonged or shortened, depending on whether the defect for the fork used is caused by the fixation of the stapes or by changes in the structures in the cochlea. When these facts are kept in mind we can readily understand the explanation of such results as a prolongation of bone conduction for the A fork and at the same time a shortening of bone conduction and a positive Rinne for the a^1 fork in the same case, a result not infrequently met with.

We come now to the third general group of otosclerotic cases, those cases where the foot-plate of the stapes has remained free, and where the spongifying of the capsule of the cochlea has produced more or less extensive nerve deafness.

Siebenmann was the first to report the postmortem findings in such a case where the functional tests indicated nerve deafness, and where foci of spongifying in the capsule of the cochlea established the diagnosis of otosclerosis.

The diagnosis of these cases from the other causes of nerve deafness cannot, with our present knowledge, usually be made. In some of these cases, however, the diagnosis of otosclerosis can still be made with a reasonable degree of certainty. One such case I presented before the Chicago

Laryngological and Otological Society in 1903.[1] This case was briefly as follows: A young man, 27 years old, a university student, general health good, no previous general illness, and no history of syphilis, began three years previous to detect insidious development of tinnitus aurium and defect in hearing, first in one ear, but after a few months involving both ears. There was complete absence of any evidence of middle ear catarrh. The membrana tympani in both ears was quite normal, but showed in a most characteristic manner the reddish glow transmitted from the wall of the promontory, which Schwartze first pointed out as characteristic of otosclerosis. Here we have a history with symptoms and physical findings typical of otosclerosis yet the functional tests discovered the findings typical of an involvement of the inner ear. They were as follows: Whispered voice heard in the right ear only when close to the meatus, in the left ear at six inches from meatus. The Weber test lateralized in the left or better hearing ear. The duration for bone conduction was markedly shortened and the Rinne was positive. The a^1 fork (64 d.v.) was heard by air conduction normally in both ears, while the hearing for the c^4 fork was markedly shortened. From the history of this case with the physical findings and the results of the functional tests I believe the diagnosis of otosclerosis was justified, the spongifying process having involved extensively the basal coil and even the wall of the promontory, as shown by the presence of Schwartze's symptom, producing the typical picture of nerve deafness, but leaving, up to that time, the foot-plate of the stapes free, and therefore giving none of the results of obstruction in the conducting mechanism so characteristic of the usual case of otosclerosis.

I have studied another case this past year where the functional examination shows an extensive involvement of the cochlea, and yet where the absence of any constitutional disease that is known to produce labyrinthine deafness, and the fact that the process was an insidious one, coming on before the age of 20, when taken with the fact that a brother had developed the same type of deafness, coming on in exactly the same way, led me to believe that we had probably to deal with otosclerosis in which the basal coil of the cochlea was involved without producing any interference with the foot-plate of the stapes.[2]

The diagnosis of otosclerosis with fixation of the stapes from deafness due to middle ear disease can, as a rule, be readily made. Whenever there is found a normal drum membrane and Eustachian tube in a case of insidious development of deafness where the functional tests show an interference with the conducting mechanism, such as a prolongation of bone conduction, a negative Rinne, and a decided elevation of the lower tone limit, the diagnosis of otosclerosis is justified.

In the incipient cases of otosclerosis, where the defect in hearing is still only slightly developed, the question will sometimes arise whether this defect might not be due to an adhesive middle ear catarrh in which the alteration of the drum membrane is so slight as to be scarcely discernible. Yet in these incipient cases the diagnosis of otosclerosis can often still be made with a reasonable degree of certainty by finding the evidences of obstruction in the conducting mechanism, the prolongation of bone conduction and the elevation of the lower tone limit, developed out of proportion to the degree of deafness.

It is often claimed that a diagnosis of otosclerosis in these incipient cases can be made by noting the absence of improvement in hearing after inflation, whereas in beginning deafness due to catarrh of the middle ear, considerable improvement after inflation is usually noted. This test cannot always be relied upon, as one occasionally finds considerable improvement in hearing after inflation in these cases of beginning otosclerosis, where the foot-plate of the stapes is still apparently not firmly fixed by ankylosis. I have under observation now a young woman who consulted me a few years ago because of beginning deafness and tinnitus aurium. After making an examination a diagnosis of incipient otosclerosis was made, yet in this case, after half a dozen inflations with massage of the ossicles, the hearing improved for the whispered voice from 2 feet to 9 feet in the one ear affected at that time. The subsequent history has shown conclusively that the diagnosis of otosclerosis was correct, although the improvement in hearing continued for the greater part of a year before the progress in the condition overcame the temporary loosening up of the stapes thus brought about.

Still more difficult of diagnosis than these incipient cases are those cases where an otosclerosis develops on top of some previous middle ear disease. Anything like a positive diagnosis in such cases is usually out of the question. Still otosclerosis may be suspected in spite of the presence of alterations in the membrana tympani where the development of the deafness is an insidious one, coming on in early life, and where the functional tests show in proportion to the degree of deafness an exaggerated prolongation of the bone conduction and negative Rinne, together with a marked elevation of the threshold of hearing for the lower tones and a well-developed defect for the higher notes of the Galton whistle. The diagnosis of otosclerosis will be all the more certain in such cases when the history shows other cases of deafness in the same family coming on insidiously in middle life and resulting finally in profound loss of hearing.

With the knowledge that the process known as otosclerosis or spongifying of the capsule of the labyrinth is by no means restricted to producing bony ankylosis of the footplate of the stapes, but that there occurs at times a more or less extensive involvement of the capsule of the cochlea, producing symptoms of nerve deafness, as well as of deafness due to fixation of the stapes, the problem of diagnosis becomes at times one of the most difficult in the whole field of otology.

On the other hand, as I have attempted to bring out in this paper, with the knowledge we now have of the pathology of this condition, the manner of onset, and the associated clinical symptoms, the diagnosis of otosclerosis not only from nerve deafness, but from other conditions producing obstruction in the conducting mechanism, can usually be made with all the positiveness which any problem in diagnosis permits.

REFERENCES.

1. Otosclerosis or Spongifying of the Capsule of the Labyrinth. Annals of Otology, Rhinology and Laryngology, March, 1903.

2. A Case of Labyrinthine Deafness with "Tone Islands." The Laryngoscope, Vol. xviii, p. 343, May, 1908.

XXVII.

THE NON-ACOUSTIC FUNCTIONS OF THE LABYRINTH: A REVIEW OF OUR PRESENT KNOWLEDGE OF THE ORGANS OF STATIC AND DYNAMIC EQUILIBRIUM.*

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In presenting to this body a compilation and abstract of the work of others in the form of an historical review and a formulation of our present knowledge of the function of the inner ear in relation to equilibrium and orientation, I feel the requirement of justification. My plea is the subject itself, which is one of increasing interest and growing practical importance. Incomplete and hasty as the review must be in the time at my disposal, a sifting of the evidence and a summing up from the standpoint of practical otology may not be without value.

The history of systematic and scientific investigation of the physiology of the inner ear and especially of the complex function of equilibrium dates back for over a century. The questions involved are so intimately related to other chapters of physiology, notably of the visual perceptions, the sense of space, direction and position, and they are so often suggested by the practical experience of the clinic and operating room, that workers in the most varied fields have contributed to our present knowledge. We have the exact and careful animal experiments of Purkinje, Flourens, Goltz, Ewald, Breuer, and many others; the clinical observations of Lucae, Bezold, Meniére; the operative findings of Schwartze, Jansen, Hinsberg, Passow, Kipp and Burnett; the neurologic investigations of Bechterew and Baginski; the patient elaboration of tests by v. Stein, Barany, Krotoschiner, Koranyi; the studies of the functional defects of deaf-mutes by James, Bezold, Kreidl, Mygind, and the psychologic applications and analyses of Cyon, Muensterberg and Warren, while in the important field of visual equilibration we are indebted to Helmholtz, Hering, Aubert, Delage and a host of physiologists for detailed tests of normal reactions and an arrangement of the varied data on the basis of uniform and simple principles.

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*Read at the annual meeting of the American Otological Society, June 23-24, 1908.

The writer has made free use of the chapters on equilibrium and functional tests in W. Nagel's Handbuch der Physiologie des Menschen, Vol. v, 1905, and Geo. Boeninghaus' Lehrbuch der Ohrenheilkunde, Berlin, 1908, many passages being cited verbatim, especially from the last named excellent treatise. The anatomic details follow Schwalbe, Anatomie der Sinnes-Organe, in Bardeleben's Handbuch, Jena, 1905, with modifications after recent writers.

Within the last ten years the literature in this wide field has become so voluminous that its mere citation would be impossible. It follows of necessity that many investigations have been omitted and others barely mentioned in my review. I have endeavored, however, to do justice to the subject by paying most attention to fundamental advances both in theory and practice and to the work of those investigators whose original experiments and observations have been most valuable.

The advance of clinical otology and particularly of its surgical branch has of late been striking and gratifying. One of the most important conquests has been the systematization of operative indications and technic in the surgical diseases of the middle and inner ear and their not infrequent intracranial complications. Many theoretical questions in diagnosis and symptomatology have been answered, and many anatomic minutiae learned, by the findings at operation and by the results, negative and positive, of surgical intervention. For the purposes of the aural surgeon the possibility of the application of all symptom interpretation, and of all diagnostic methods will depend in the last analysis on the degree to which they conform with and explain the manifestations of disease in actual practice. While we must admit this without reserve, there can, I think, be no doubt of the value of continued study of function and its disturbance from the experimental theoretic side, no doubt that an elaboration and critical analysis of all rationally scientific methods will be of eventual service to the practitioner, and that the final result of accurate and painstaking investigations in laboratory and clinic will bring theory and practice into much desired accord and correspondence. The study of the functions of the inner ear and the systematization of functional tests is by no means complete, but enough has been done to show that we are moving in the right direction. The objections which have been raised to the errors inherent in this or that test, the exceptions to minor points of this or that theory are details, not fundamental, and cannot invalidate the principles involved nor prevent their eventual application. In fact, the practical objections of competent clinicians and surgeons can only serve a good purpose in correcting errors while they stimulate anew the search for the truth.

BIOLOGIC BASIS OF EQUILIBRIUM, TROPISMS.

A study of that complex of sensations and reflexes, of inhibitions and innervations, of conscious adjustment and automatic correction which is summed up and comprised in the term equilibrium, may be facilitated by reference to the simpler conditions existing at a lower level of structural and functional development. Biology has shown that there is present, and possibly inherent, in all forms of multicellular life, a tendency to show definite, and apparently purposeful reactions, so-called somatic reactions, to a large number of cosmic influences. In contradistinction to the visceral reflexes, which have to do with the internal organs and their functional response to stimuli in the form of such acts as swallowing, breathing and so on, these so-called somatic reactions consist in changes of position or attitude and may be produced by a number of agencies. As these agencies evidently act as stimuli, and the response motion has the effect of altering the relation of the position of the organism to the source of stimulation, these changes have been grouped under the title of "tropisms," or turnings in a wider sense, and meaning not so much a rotation as a movement toward or away from the source of irritation. It has been shown, notably by Loeb and his school, that there is a uniform tendency to react to darkness and light by somatic alterations (heliotropism); to gravitation (geotropisms), to electrical current (galvanotropism), and to heat as well as to mechanically produced air vibrations (rhythmotropism). The varied changes of attitude and position can all be reduced, teleologically, to a simple formula or principle, the gist of which is that a final optimum of position is secured for the organism. This optimum may differ for various genera, species, and even individuals, but it is generally characteristic and imposed by the needs of the particular class, and by the desiderata of equal symmetrical innervation and a maximum saving of sensory as well as motor energy.

With increased complexity of structure and a differentiation of the body form into head and trunk, upper and lower, or abdominal and dorsal aspects, the somatic reactions to gravity become more and more accurate and delicate, and finally produce characteristic positions of balance and equilibrium both at rest (static) and in motion (dynamic). In higher animals, these automatic adjustments are found to be influenced by volition and as the cerebral hemispheres develop in the ascending series of animals, phylogenetically, there is evident an ever increasing domination, through inhibition and active innervation, by the will and by intellect. The highest stage of this domination, notably in man, includes the recognition of a summation of subjective sensations of an ego, as opposed to an objective something outside produced by a systematized externalization of perceptions. This process it is which finally produces the conception-complex of ideal space or direction to which we unconsciously refer, and with which we compare our motions and sensations. Association, memory and habit combine to produce nerve paths of least resistance and to produce in sensory organs and cerebral centers so-called local signs, which may be defined briefly as definite conceptions of the relation of external agencies as cause, with subjective sensations as effect. Thus we have retinal local signs corresponding to different eye positions, and to different degrees of innervation of the ocular muscles, to accommodative states and to various points in space; cerebral local signs for all these data and for all of the varied sensory perceptions.

FACTORS OF EQUILIBRIUM.

The adjustment of the individual to space, equilibrium, and his recognition of that adjustment, orientation, is, in man at least, a complex function. Touch and sight, muscle sense, in its widest meaning, sensations of pressure or tension, of lightness or heaviness, in tendons, joints and viscera, as well as the feelings produced by consciousness of changes or states of hyperemia and anemia, all play a part. Nagel has shown that many of these agencies act so automatically that they may not enter fully into our consciousness, and special adaptations of conditions are required to exclude one or the other in the attempt to analyze and explain a single one. In fact it has been questioned whether this function of balance is a sense at all and whether it is not rather to be considered as an unconscious judgment-reaction of the sentient organism to the sum of sensory impressions. This question is of psychologic rather than practical importance, and it has been found possible to make a fairly accurate and complete analysis of the functions of each factor. The part played by the visual organ has been studied with special ingenuity and exactness, and the work of Helmholtz, Heryng, Aubert, Fechner and many others has finally given us a clear idea of the ocular function in space perception.

Stated very briefly and generally, there is a definite and constant reaction to visual stimuli, having as its object the production of an optimum position of the eye-ball and clear central vision of external objects by means of macular fixation. This is expressed by a tendency to preserve the relation between verticality of the corneal meridian with verticality of external objects, to compensate for inclination of these objects by an opposite inclination of the cornea, and secondarily but necessarily, of the retinal meridians. This correction is automatic and unconscious, and is complete for small degrees of inclination below 10°, that is, for alterations below the limit of perception. It is rather striking that this correction fails at a point where, as we shall see, another sensory-motor mechanism is brought into play by the mechanical conditions arising with higher degrees of inclination.

Fixation of objects at rest, a second and important factor of space perception, is a summation of all the ocular motions necessary to produce clear macular vision. The position of the fixed object is unconsciously judged not only by the stimulation-sense of the muscles engaged in fixation, but by a reference to the sense of position in our own bodies. There is here a definite interrelation of the subjective and objective, objects being referred to the known or felt position of the subject, and the position of the subject also judged, at least in part, by known positions, known that is from experience with objects in the external world. Fixation of moving objects also follows an instinctive tendency to bring them into the range of macular fixation. In the case of definite motions, that of objects progressing through space in one and the same direction, we can recognize two main divisions of eye motions. First, a rapid associated turning of the eyes toward the source of the moving objects, that is against their direction, real or supposed, for the purpose of catching that object on the macula. This motion, as has been shown by numerous experiments, is not associated with vision. In fact during this, recovery or compensation leap (the "Einstellung" of the Germans), there is no clear vision whatever. The second motion is in the direction of the moving object and consists in keeping step with it, as it were, so that it is always in the line of sharpest vision. As this motion is regularly and invariably associated with a definite form of motion in external objects, its production, by any means whatever, will produce a sensation of such motions. It has also been shown that our knowledge of subjective motion is largely dependent on the feeling of eye-muscle-motion which accompanies the change of position in external objects. Numerous illusions of motion are due to apparent changes in the position of external objects. We are all familiar with the false sensation of moving in an opposite direction when in a stationary railroad train past whose windows a train of cars is slowly moving. We are largely dependent on our eyes for our habitual sensations of motion and change of position, and when deprived of these, as in the dark, our sense of motion direction may be greatly diminished. This is particularly true of rotation-motions and the lying position. Progressive motion, forward or backward, in the erect position, is uniformly attended by sensations of resistance or friction between the body and its support. The absence of these tactile and visceral sensations, or their changed relations to our experience, accounts for the ease with which we can be misled as to the direction in which we are moving when lying down, as at night in a sleeper or on shipboard.

Besides the definite subjective sensations produced by changes of position, we note definite somatic reactions, "specialized tropisms," we might call them, having as their conscious or automatic object the restoration of balance, i. e., the regaining of the optimum position, which we may assume is normally and habitually occupied. This adjustment is in the nature of a correction or compensation. It depends upon muscle motions, largely involuntary and reflex, brought into action by sensations of lack or loss of balance. It is significant for the safety of the organism that disturbances of the motor arc of this mechanism do not vitally affect or seriously interfere with balance. Differences of muscle strength for instance, in the two sides, even marked irregularities, such as a flat foot or a sprained ankle, do not cause loss of equilibrium. There is probably unconscious adjustment for these factors, as there is for the unequal strength of right and left hand when both are used together in pulling or pushing. The sensory component, on the other hand, is of the greatest importance, as it is through the sensations of resistance that the varying degrees of muscle innervation are called into action, and as a matter of fact, disturbances of sensation, anesthesias, are particularly apt to cause confusion in somatic reaction for the preservation of equilibrium.

THEORIES OF THE FUNCTIONS OF THE LABYRINTH.

The striking anatomic peculiarities of the structures of the inner ear, the snail-shell whorls of the cochlea, the regular curves and geometric arrangement of the semicircular canals, and the suggestion of complex function and of necessary purpose afforded not only by structure but by their presence throughout a long range of the animal kingdom, attracted early attention and inspired investigation, while they seemed to hold, as it were, within themselves the answer to the riddle of their meaning. The arrangement of the canals, particularly, in three principal dimensions of space was at once felt to be of fundamental significance, and from the earliest years of modern scientific investigation we find this special relation as the starting point of theory. The most obvious connection was that with audition, and it was assumed that the three semicircular canals mediated sensations of sound coming in any of the three main directions corresponding to their axis, or analyzed atypical sound directions by proportional representation in the three dimensions. This theory crops up at intervals in the history of the investigation and has persisted until the present time. It is associated with the names of Autenrieth (1802), Cyon (1854), Brueckner (1888), Hensen (1893). Many variations were played on this theme. The function was thought to consist in a destruction or dampening of tone-waves which had already acted on the auditory organs, sensu strictiori (Czermak, 1860; Malinin, 1866), in perception of noises as opposed to tones, i. e., non-periodic vibrations (Schiff, 1857; Hasse, 1867); in perception of muscle tones produced by contraction of head muscles and thus indirectly of position of the head in space (Bornhardt, 1872); by bone conduction of external sounds (Ogston, 1869), and with the purpose of directing eyes and ear toward the source of sound (Münsterberg, 1889). A teleologic modification of this basic theory allowed the inference that a special sense of direction, acoustic stereognosis, as well as the homing instinct of animals was mediated by this appartus. Among other theories which attribute a non-acoustic function to the labyrinth we may mention those of Cyon and of Högyes, who believed that the nerve terminals in the labyrinth were intended to produce definite tetanic positions of the eye muscles, the 2x6 nerve endings being in communication with the 2x6 eye muscles for the purpose of producing unconscious eye positions which are not compensatory but reflex.

NON-ACOUSTIC THEORY (BREUER, MACH, CRUM BROWN).

The theory of a specific, equilibristic function of the labyrinth was based almost from the beginning on the results of careful experiments on animals and human beings, and accordingly progressed by slow and steady steps, rather than by leaps and bounds. Among the earliest observations were those of Purkinje (1820), on rotation and vertigo, and those of Flourens (1825) on experimental section of the horizontal semicircular canals. The latter investigator found in doves and rabbits that hearing was not affected, but that there was a typical disturbance of equilibrium, with characteristic compensatory movements of the head and body. Section of the other canals caused similar pendulum motions of the head in the axis of the injured canal. Nearly fifty years later, Fr. Goltz, in Strassburg, took up the forgotten experiments of Flourens and repeated and extended them, showing that the compensatory motions were not forced motions and did not depend on auditory sensations, but were specific sensory reactions. The semicircular canals are the sense-organ for the equilibrium of the head and mediately for that of the entire body. The adequate stimulus is the gravitational pressure of the column of endolymph. A few years later, Breuer and Mach, in Germany, and Crum Brown, in England, suggested a modification of the theory of Goltz. The first named author called attention to the fact that the mechanical requirements of this theory could only be satisfied if the semicircular canals were suspended in a medium like air, of a specific gravity appreciably lighter than that of the endolymph, whereas, in fact, they are surrounded by perilymph which is of about the same density. The modification of Breuer and Mach consisted in the hypothesis of endolymph motion in place of endolymph weight-stress as an adequate stimulus, and this slight change was the cornerstone of a new structure. As the theory of Breuer and Mach with its adaptations to advances in experimental and clinical investigation is the most complete and satisfactory at the present time, and as most of the clinical tests and diagnostic conclusions are based on acceptance of its fundamental assumptions, somewhat more detailed consideration of this theory may be advisable.

PLAN OF SEMICIRCULAR CANAL SYSTEM.

I shall not tax your patience with a description of the anatomy of the labyrinth further than to call attention to the fact that we have to deal with a bilateral, symmetrical structure. Each canal of a side corresponds in plane to a canal of the opposite side; thus the two external canals in the horizontal plane, the anterior or superior canal of one side with the posterior or inferior of the other side. (Fig. 1.) It will also be noticed that the ampullar bulbs of corresponding canals are at opposite ends; the ampulla of the right horizontal being on the right, while that of the contralateral canal is on the left; the ampulla of the right superior canal being in front, above, and that of its synergist, the left inferior, being behind, below. Furthermore, the greater part of each canal is posterior to the ampulla, so that bearing in mind that the entire system is about on a level antero-posteriorly with the axis of rotation of the head, any rotational change will correspond to an opposite rotation of the corresponding canal. The practical significance of this arrangement will be pointed out in the analysis of the functional mechanism of the labyrinth.

ANATOMY OF THE LABYRINTH.

The membranous labyrinth (Fig. 2) containing the terminals of the vestibular branch of the acoustic nerve is enclosed in a bony capsule, the osseous labyrinth, occupying the middle of the petrous portion of the temporal bone. In animals of the higher vertebrate class the structure is very like that in man. The sagittal axis of the system is directed inward at an angle of about 45°, and the horizontal canal is not in a horizontal plane, but inclined backward at about the same angle.* The posterior crura of the two vertical canals on either side join and enter the utriculus by a common port, so that there are only five openings instead of six. Each canal has a spherical enlargement, the ampulla, at one end. Within this ampulla we find a prominence so marked as almost to occupy the central axis of the canal. On this crista we find sensory epithelium with long pliable hairs, not floating free in the endolyymph, being held together by a gelatinous mass, the cupula terminalis. At two points in the membranous labyrinth we find, respectively, a vertical and a horizontal collection of sensory epithelium, very similar in arrangement and structure to that of the ampullae, the macula utriculi and macula sacculi. The hairs are shorter and stiffer than those of the ampullae, are held together by a stiffer mass (otoconium), which, however is not gelatinous, but composed of the so-called 'otoliths ("statoliths" according to Verworn), microscopic crystals of carbonate of calcium. The rest of the wall of the membranous labyrinth has no nerve terminals, but gelatinous threads have recently been traced by Breuer from the wall of the utriculus to a drop-like accumulation of the same material on the otolith mass which they evidently nourish and renew. The plane of the macula utriculi corresponds to that of the external semicircular canal, i. e., in the upright position it is inclined backward at about 45°. The otolith plate of the sacculus, under similar conditions, runs at about the same angle from above and behind, forward and downward, in the anterior posterior plane, i. e., parallel on either side. In lower animals there is a third macula, the lagena, allowing vertical displacement of the otolith plate, while both utricular and saccular macula are horizontal, but at right angles.

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*This special arrangement of the so-called horizontal canal and of the maculae would indicate that the erect position of man has not existed long enough to influence these structures of the inner ear. They are still in the same position as those of the "beasts of the field which creep upon the earth."

NERVE PATHS AND CEREBRAL CONTROL.

The vestibular nerve (Fig. 3) starts from the ampullae. one branch (ramus medius N. viii) from the posterior ampulla going separately through the foramen singulare near the meatus internus, another from the macula sacculi, superior ampulla, and the external ampulla (first branch of ramus superior), through the upper portion of the terminal plate, and a third, utricular branch (second branch of ramus superior), through its lower portion, through the vestibular, bipolar ganglion, which according to Hensen is the neurologic nerve terminal of the vestibular branch. The vestibular nerve now enters the pons and reaches the dorsal nucleus of the eighth nerve (Deiter's and Bechterew's nuclei). Here the peripheral neuron ends. The central filaments can be traced across around the fourth ventricle and the tegmentum or roof to the tegmental nucleus, most of the fibers going to the opposite side. This path brings the vestibular nerve into relation with the anterior columns of the cord and with the abducens. There is no known connection with the cerebral cortex. The cerebellar cortex is intelligible as an indicator of degree of tonus, contraction and effort, in various body muscles, notably those of eye and lower extremities, established voluntarily by the brain, or by reflex from tactile, muscle, visual and balance sense, to preserve body equilibrium and possibly reflex fixation of external motion. In time it would become, as we know of the cerebral cortex, a store house for memory-pictures of sensation-states connected with eye-motion, tactile impressions and muscle tonus, in their relation to balance and the perception of motion, direction, rotation and the conception of ideal space, and would thus aid in regulating subjective sensations by referring them to a common standard of tradition, experience or memory. A discrepancy of signals received at a given time, whether from eye, tactile or muscle sense, or balance centers in labyrinth, either with each other or with memory standards, would produce the effect of a contrary innervation and the markedly unpleasant subjective sensation of confusion which we call dizziness. The unconscious judgment factor in complex sensation of balance and its disturbance in dizziness are shown by the gradual development and increase during life of the capacity for both, the susceptibility of highly organized and trained intellects to dizziness, the comparative immunity of children and stolid, phlegmatic, illiterates, and the ease with which the sensation of disturbed balance or dizziness can be elicited by slight modifications and confusions of visual impressions. This is particularly the case with the swinging mirror which produces almost immediately a feeling of marked vertigo and even nausea.

The labyrinth is in connection with the ocular muscles presiding over abduction to the same side, i. e., external rectus of the corresponding eye and internal rectus of the opposite eye; the occipital muscles and trunk muscles of the same side and with lateral rotators of the head to the opposite side (Marikovszky, 1904, rabbits). The connection with the oculomotor nuclei and the anterior horns of the cord runs through Deiter's nucleus (Adler), and the central end station for centripetal sensory stimuli is situated in the cerebellum. According to Stefani, the cerebellum is a central organ whose activity is principally stimulated and maintained by impulses emanating from the labyrinth and whose function consists in the preservation of muscular tonus which is required for the maintenance of a stable body position in normal equilibrium. The cerebellar cortex is not to be considered as the center for sensations of motion and position (Lange, Nagel). The labyrinth, besides its function in maintaining tonus, mediates these sensations and it is to be assumed that they have a representation in some cortical area, but this area has not yet been determined.

THE MECHANICS OF LABYRINTHINE FUNCTION ACCORDING TO BREUER AND MACH.

(Figure 4.)

With every rotation of the head the endolymph contents in the semicircular canals lags behind on account of inertia, so that in the canal corresponding to the plane of rotation there is a contrary current or pressure stress of lymph, which will produce torsion on the terminal plate and the cilia of the sensory epithelium projecting into the lumen from the ampulla, and, it is to be assumed, cause a specific irritation of the nerve terminals. The axis of head-rotation will determine in which associated pair of canals this endolymph motion will occur. In motions which are not exactly in the axis of any pair of canals, there will be an associated action of several pairs. It is evident that the physical conditions for a contrary current due to inertia are satisfied only as long as there is an acceleration of rotation of a whole canal. After uniform rotation has been instituted there can be no relative motion or disturbance of balance between contents, endolymph and canal wall. Every positive or negative acceleration, however, and particularly a sudden cessation of rotation, must produce a marked displacement of the column of endolymph, which will continue to move in the direction of previous rotation (remnant of motion). The only serious objection to this hypothesis is the extremely small caliber of the canals, which in man are only 0.1 mm. square. This has been met by the substitution, for actual motion of the endolymph, which would probably be neutralized by friction against so narrow a wall, of a pressure state in the lymph such as might produce distortion in a vertical membrane hung up in its course. The crista projecting into the lumen of the canal would represent such a membrane. (Fig. 5.)

Breuer's theory of the static function of the labyrinth, as applied to sensations of position, is based on the assumption that the otolith mass on the macula is of so much greater density than the endolymph that various head positions must produce various degress of pressure or stretching in the sensory hairs. With a macula in the horizontal position the macula would press directly on the hairs which carry it. In the reversed position, with the otolith plate below, there would be traction on the hairs. In any intermediate position of the system there must be distortion of the hairs which would reach a maximum, say, when the plane of the otolith plate and that of the macula were vertical in space. The obvious and striking difference of this mechanism from that of the ampullae and canals lies in a persistence of the mechanical conditions and of the torsional stimulus induced by them, as long as the position of the system in space is unchanged, while the mechanical influence on the cupula in the ampulla takes place only when there is a change in the rate of motion of the labyrinthine contents. This duplicity of function, or rather of excitation modus, will serve to explain the distinction between those sensations and reflexes which are associated with motions, passive or active, of the head, and those which are due to permanent positions of the head in space.

Experiments on lower animals in regard to the function of the otoliths or otocysts, which can be removed without injury to other parts much more easily than the semicircular canals, show that they are essential for the maintenance of equilibrium. Crabs, squids, marine snails and other animals deprived of these organs, swim on one side or on the back, or in circles. These experiments with defects are supplemented by one very instructive series of irritation tests. Certain crabs shed their sand otoliths at definite periods. Kreidl let crabs renew their otoliths with iron filings and then found that the approach of a magnet from one side was followed by a tilting of the animals to the opposite side, evidently for the purpose of restoring the balance which in sensation at least, had been disturbed by the displacement of the magnetic otolith.

The position of both horizontal (utriculus) and vertical (sacculus) otolith plate is quite logically explained to serve in erect and in supine position, respectively, the former in a state of functional equilibrium when the body is erect, the second, under stress, and vice versa.

Otoliths produce sensation of position, requiring steady and prolonged strain of gravity, and are not easily set in action by the usual degrees of motion-acceleration to which we are subjected. They would be useless if they reacted to every such impulse, as we should have subjective sensations of change of balance in every progressive motion. A forward jump would produce torsion of horizontal plate corresponding to falling backward. As a matter of fact such sensations occur only when rotational motions are combined with action of gravity, illusion as to real vertical inclination of body, and judgment of slant in surrounding objects corresponding to the resultant of gravitational and centrifugal factors..

There are certain positional reflexes in distinction to rotational reflexes, the most important of which are the compensatory positions of the eyes which occur when the head is tilted and which persist as long as this head position is maintained. A similar reaction is noted in birds and reptiles which keep the head in a certain position, in relation to the vertical, no matter how the body is pointed. These reflexes are explained by the function of the otoliths and their dependence on gravitation. As a certain deviation from the vertical is required before gravitation can produce torsion on the sensory hairs of the otoliths, we can now understand the function, mentioned above, of the retinal meridians and of compensatory inclination in slight deviations. With an increased angle of tilt, the otoliths are affected and the change in head position becomes apparent to consciousness. Tests of tilting at various angles support this view. When titled backward gradually, we underestimate the angle until 50° or so has been reached. From there on, there is an increase in the subjective sensation of tilting so that with a backward inclination of .75° we think we are in a horizontal position. From this point on the sensation of tilting increases rapidly, and when tilted so that the head points downward 15° we have the feeling of being inclined 45°, and think we are standing on our head when it is in reality only 30° below the horizontal.

THE EXPLANATION OF SENSORY PHENOMENA AND SOMATIC REACTIONS ON THE BASIS OF THE BREUER-MACH THEORY.

(Figure 6.)

The Phenomena of Rotation.—At the beginning of a rotation motion, and with every change in its acceleration there is a definite sensation which is correctly interpreted, but this sensation disappears as soon as the rotation has become uniform (unless a change is produced by alteration of the position of the head). Retardation of the motion is interpreted by sensation as a motion in the opposite direction, and this is noted even after all motion has ceased. This subjective sensation of false motion, Mach's rotational vertigo, is explained by the motion-remnant in the endolymph, which is now active in an opposite direction to that caused while the system was in actual motion. The sudden sensation of motion coming on during uniform rotation, on turning the head actively through 90°, is also explained by a temporary acceleration imparted to the endolymph.

Reactions similar to those observed on passive rotation have been noted after irritation of a semicircular canal in which motion in the endolymph was produced by stroking or some similar mechanism (Ewald). There is invariably a turning of the head and a contrary motion of the eyes in the axis of the irritated canal (irritation test). Similar reactions were noted in ear operations when a semicircular canal was exposed (Jansen). In doves, the head was turned to the sound side if the fluid was displaced toward the ampulla, and the irritation reaction was more marked than when the fluid moved in the opposite direction, i. e., from the ampulla, when the head turning was toward the irritated side. We noted above that the ampullae are at opposite ends of each pair of canals. Bringing this into connection with the data gained by animal experiments we can understand that rotation to the right, for example, will produce a sensation of turning in this direction, the endolymph moving in the right horizontal canal from left to right toward the ampulla, as the occiput and with it the backward arched semicircular canals move from right to left. In the left semicircular canal the movement of the endolymph, in the same direction, i. e., from left to right, will be away from the ampulla and produce a minimum of irritation and, accordingly, of sensation. In left rotation we shall have a corresponding predominance of sensation in the left horizontal canal. If both sides are functionating normally there will be a correct interpretation of motion, and uniform somatic reaction on turning in either direction. If, on the other hand, there is an increased sensibility in one, this will be associated with intensified reaction. Again, destruction of function will allow the normally subordinate reaction from the other side, to which we have just referred, to become evident, and the weak sensation and reaction from the contralateral canal, produced by motion of endolymph away from the ampulla, will produce a positive reaction. As we shall see later, this has been made use of for a separate test of the labyrinthine function of the two sides.

DESCRIPTION OF NYSTAGMUS MODEL.

(Figure 7.)

The quadrant is made of thin tin through which nails are driven at regular intervals to represent objects fixed during rotation. The nails are covered with thin rubber tubing to deaden the shock and noise, and a paper indicator stuck in the tube to show from a distance. A piece of rubber tubing is slipped over the pointer, too, to allow variation and adjustment of length, as in the "abducted" position, it does not project over the edge of the turn table as far as in normal position, and so fails to strike the nails on the quadrant.

When properly adjusted, with pointer normal, each rotation alteration of the turn table produces a deflection of the pointer, which is caught and carried backward by the peg moving in the opposite direction. When the pointer has been carried along for a space, it slips from the nail, or is released from fixation and jumps to the next object, or nail, with a rapid motion which is in the direction of actual rotation, i. e., toward the source of apparently or actually moving objects. This adjustment, or rapid correction leap, the "Einstellung" of the Germans, is the more marked of the two motions in nystagmus, and it is according to the direction of this "rapid" clonic motion that the nystagmus is characterized as right, left, and so on. It has been shown that there is no clear vision during this rapid compensation or adjustment motion, and this may be seen quite clearly when we read lines of print. There is an adjustment after reaching the end of each line, the eyes jumping back in order to fix the first word at the beginning of the next line, far to the left. During this leap we do not see a single word clearly, and only one or two letters are picked up dimly.

Rotation of the disc in either direction produces analogous results, i. e., nystagmus toward the direction of rotation. The effect of abduction, as by active fixation to one side, during rotation, can also be demonstrated on this model. If we attach a third elastic band (el_2) to the pointer so that it no longer points out radially from the center of the turntable, but, as shown by the dotted lines, is inclined toward one end of the line of nails on the quadrant, we find that the catching and release of the pointer, which as explained above corresponds to fixation-tonus and compensation-leap, are very slight when the pointer represents an eye looking backward, i. e., away from the direction of rotation, and very marked when the eye looks forward in the direction of turning. This is not only in harmony with the observations of every day life and clinical study, but can be very simply explained mechanically and visually. On looking backward away from the source of motion there is less apparent motion; the object moves through a smaller arc in the given time. If we look directly backward, for instance, there will be practically no motion, the object merely becoming "small by degrees and beautifully less." On looking forward in rotation the objects come into the field of vision sooner and are in the field of fixation longer; there is a corresponding sense of greater motion and a greater actual leap of compensation or clonic fixation; greater evident nystagmus. Mechanically we must remember that in looking forward in right rotation for instance, we consciously innervate the muscles for right abduction, so that this volitional stimulus would be added to the unconscious or automatic adjustment of fixation, produced by motion of external objects, which, as we have seen, also corresponds to abduction in the direction of rotation. In looking away from the rotation to the left, we innervate an opposite set of muscles consciously, to those which are being set in action automatically, and the two impulses are not summated as before, but tend to neutralize each other. Clinically it has been noted that nystagmus is increased on looking toward the diseased side, i. e., in the direction of apparent rotation, and that it may be apparent only in forced abduction to this side. Conversely, nystagmus may be inhibited by looking toward the sound side. This abduction also tends to inhibit or entirely prevent vertigo, and is made use of for this purpose by ballet dancers and skaters to prevent dizziness in whirling. The head and eyes are turned backward, away from the direction as far as possible, and forced fixation in opposite abduction used to keep the eyes in line with one point, usually the center of the auditorium. In this way the motion of the head and eyes is artificially slowed and reduced to a minimum. After the body has moved through a large arc and made almost a complete revolution, the head is, actively, turned to catch up with it, and fixation to one side again takes place. In this way we have an interrupted motion of the head and eyes, giving the semicircular canals a chance to come to rest frequently and producing a small repeated reaction with which the sense of balance can cope without sensation of dizziness, in preference to allowing an accumulation of irritation through prolonged endolymph rotation, which on cessation of whirling, might produce extreme giddiness and loss of balance.

TESTS OF THE STATIC FUNCTION.

It has already been stated that the adequate stimulus for the ampullary organs is the motion of the endolymph produced by rotation. As in other sensory organs, a number of other stimuli can be made to replace the normal and physiologic irritation. Thus, condensation or rarefaction of air in the external auditory canal, especially when the external horizontal canal is exposed, will produce a change in the intracanalicular lymph stress. Even concentrated light under these conditions, will act as an irritant (Lucae). Mechanical pressure by a probe or by granulations, on the oval window will act similiarly, and thermic irritation by hot and cold water or galvanic currents, will supply sufficient stimuli. In all these cases we have definite sensations of rotation motion, and definite somatic reactions of eyes and head. The manifold and somewhat complicated phenomena may be most simply summed up as follows. Each irritation of a semicircular canal corresponds to a sensation of motion which is interpreted as taking place in the direction of the endolymph stream, i. e., as a rotation toward the irritated side. The somatic reaction consists in an attempt to fix objects in motion coming from the irritated side, as they naturally would if there were actual motion. There will be a rapid turning of eyes to the irritated side, "Einstellung," with a slower fixation tonus in the direction of the moving objects, i. e., nystagmus to the irritated side. The head will turn against the fancied motion in an attempt to correct the subjective sensation of displacement, and the trunk muscles on the irritated side will receive a stronger innervation or an increased tonus for the same purpose, i. e., to correct fancied motion change or compensate for apparent alteration of balance. We shall now consider the various methods of stimulating the ampullar apparatus and note the underlying principle which will explain the unifomity of reaction.

Dizziness and quite possibly nystagmus (although this has not been studied) may be caused by irritation of semicircular canals by molecular vibration of endolymph due to sound waves. The tone vibration must be of unusual amplitude to cause this phenomenon, and accordingly we note unpleasant subjective sensation of dizziness with unusually low and strong notes, as the deep contra-bass of an organ, a low pitched fog horn or the hum of a locomotive exhaust.

Physiologists have not decided whether the disturbances of equilibrium noted after destruction of the labyrinth are due to this destruction directly, say by irritation of the nerve trunk, or whether these disturbances are the expression of a predominance of the contralateral sound labyrinth and a consequent alteration of the balance of sensory perceptions in the static central apparatus situated in the cerebellum. In other words, whether we have to deal primarily wth phenomena of irritation or of paralysis, loss of function. Clinically this is not of practical importance as in labyrinthine disease we have almost always an extended period of irritation due to inflammation or hemorrhage. In patients we shall have to distinguish a stage of irritation and a stage of function loss which is secondary, i. e., develops later. A sharp differentiation is not always possible as loss of function may begin while irritation persists, just as in affections of the cochlear branch there may be loss of hearing with subjective tinnitus.

The main symptoms of irritation are nystagmus, disturbance of equilibrium and vertigo, with nausea and vomiting. Boenninghaus claims that the first three symptoms are related, inasmuch as nystagmus causes vertigo or false sense of rotation, and that this in turn produces loss of equilibrium. Nystagmus, involuntary eye motion, produces a sensation of objective motion when the eyes are open or of subjective motion when they are closed; pseudo-rotation or vertigo as opposed to vertigo or dizziness in the wider sense such as visual vertigo, and so forth. The dependence of rotational vertigo on nystagmus has been demonstrated by Barany who showed that vertigo does not occur if the nystagmus is suppressed, e. g., after rotation with closed eyes, if the gaze is directed away from the direction of rotation. The direction of apparent rotation is in the direction of nystagmus, but the body, to compensate for the supposed displacement, deviates in the opposite direction. This involuntary compensatory reaction disturbs the balance of the body and leads to swaying, reeling, stumbling or falling to one side or the other.

Function loss symptoms consist in disturbances of balance without vertigo, and nystagmus without nausea and vomiting. They are not markedly different from the analogous irritative symptoms except that the latter are more marked, varied and irregular in intensity, in accordance with the varying degree of irritation.

In tests of the static function, nystagmus and balance disturbance offer objective evidence and are therefore more valuable than the subjective sensation of vertigo, which furthermore may be complicated with disturbances of general consciousness or indeterminate psychical reactions. Nystagmus is particularly valuable as a symptom (Barany) because it is absolutely independent of physical control.

CLINICAL TESTS FOR NYSTAGMUS.

Labyrinthine Nystagmus.—Irritation of a single semicircular canal in animals causes a definite motion of the eyes. If the irritation continues, a clonic spasm takes the place of the single muscular contraction. After destruction of the labyrinth there is neither nystagmus nor vertigo on rotation. A similar condition is noted in deaf-mutes with loss of both labyrinths. This form of nystagmus can be distinguished from nystagmus due to amblyopia. The latter is even, tremor-like, the former irregular, clonic, like the twitching motion seen in paresis of the external rectus muscle when an attempt is made to fix in abduction. In the usual erect position with the head held vertically, there is a horizontal or circular motion of both eyes to the right or left.

The two motions are not equally rapid, and the direction of the rapid motion determines the characterization of the nystagmus, as nystagmus to the right, left, and so on. The nystagmus is increased by looking in the "rapid direction." This can be explained mechanically by the fact that voluntary innervation now assists the subconscious innervation action of the muscles turning the eye in the direction of the gaze which is identical with the direction of the nystagmus. It is also possible that a greater arc of motion is offered for the compensatory effort as well as for the steady fixation component and that the sensation of external motion is more extended when the eyes are turned toward the source of apparently moving objects. This is undoubtedly the case in the reflex nystagmus which occurs when we fix objects from a moving train or a caroussel. The apparent motion of these objects and the visible nystagmus are both much less marked when we look back along the line and away from the direction of turning. Labyrinthine nystagmus may be so slight as to appear only in forced abduction in the "rapid" direction, i. e., toward the irritated side. The excursions may be large or small, rapid or slow. At times they can be seen only with good illumination or with a magnifying glass, on close inspection while the patient fixes an object held far to one side. Labyrinthine nystagmus may be elicited by a number of stimuli or it may be spontaneous. It is then a symptom of labyrinthine irritation, and is accordingly directed toward the diseased side. It may be assited by holding the head low and thus causing labyrinthine hyperemia. In labyrinthine irritation, nystagmus may be caused by simple turning of the head, which is quite unusual in health.

NYSTAGMUS AFTER BODY TURNING. (ROTATIONAL NYSTAGMUS.)

Active or passive rotation of the body about its longitudinal axis causes nystagmus in the direction of turning (Purkinje, 1825). Sudden cessation of rotatory motion is followed by reversal of the direction of the nystagmus, after-nystagmus. In labyrinthine irritation, rotation toward the diseased side may produce nystagmus after only one or two turns which also lasts longer than nystagmus following rotation in the opposite direction. The semicircular canal around which we turn is always irritated most actively, as under these conditions the motion of endolymph is toward the terminal nerves in the ampulla. Unilateral loss of labyrinthine function does not produce any lasting alteration of the nystagmus, but it disappears entirely.

Barany has also noted that when there is a fistula in the bony wall of the labyrinth, compression or rarefaction of the air in the external meatus will produce typical labyrinthine nystagmus and vertigo. The details of this reaction have not yet been worked out.

When both labyrinths have been destroyed, as in deaf-mutes, if only one semicircular canal is destroyed, nystagmus, will, for a time, be more to the sound side. Practically there are technical difficulties in the way of accurate observation of rotational nystagmus. It has been suggested that the observer seat himself on the turn table with the patient and palpate the latter's closed eyes with the finger tips to determine the character and direction of the nystagmus. Practically it is more simple to test the after-nystagmus which takes place on cessation of rotation. In normal individuals about ten rotations are needed to produce decided nystagmus, while in labyrinthine irritation two or three turns suffice. The subjective sensation of rotational vertigo and the disturbance of equilibrium associated with it can best be tested by having the patient get up and try to walk in a straight line after rotation has ceased.

Caloric Nystagmus.—The specific action of heat and cold was discovered in animals by Baginski in 1881, and studied and applied to human beings by Barany, 1906, who first recognized its physiologic and diagnostic significance. If the ear be irrigated with water above or below the body temperature, nystagmus is produced which is more marked as the temperature contrast increases, and ceases when water of an indifferent temperature is used. Hot water acts as an irritant, i. e., as a stimulus corresponding to rotation to the tested side, while cold water acts as a depressant, with a diminution of sensation of rotation toward the tested side, or what amounts to the same thing if the body is at rest, to a motion toward the non-tested side. Accordingly hot water nystagmus is directed to the tested side, cold water nystagmus to the non-tested side. After destruction of the labyrinth there is a loss of caloric reaction. This has been proven by tests on animals (Kubo, 1906), and observations on patients with known destruction of the labyrinth. Barany lays great stress on this point, and claims that we have in the caloric method an accurate means of determining the presence or absence of function in either labyrinth. A negative finding would mean that any objective reaction, such as spontaneous nystagmus, must be due to the labyrinth of the opposite side or to non-labyrinthine factors, such as involvement of the cerebellum, and the same author claims that the diagnosis of cerebellar abscess can be facilitated by means of this procedure. The mechanic explanation of caloric nystagmus is found (Fig. 8) in the production of currents in the semicircular canal due to the change in density of the endolymph which follows the change in temperature. Barany says: "Imagine a closed vessel filled with water at 37° C. to represent the labyrinth. Syringe one side of this cup with cold water. The water nearest this side will be cooled and thereby get a greater specific weight, tending to move toward the bottom of the cup, while on the opposite side the warm water rises. In the same way and for the same reason a movement of the endolymph is produced in the semicircular canal."

The diagram (Fig. 8) also explains sensory and motor reactions to rotation. a + shows effect of positive irritation of R. ampulla, by rotation to right, accompanied by homonymous nystagmus (R) and compensatory head turning (L). The canal rotates from left to right, producing a stress toward the ampulla (serpentine arrow at B.), and sensation of motion to R. b — shows negative or reversed stimulation, of ampulla, immediately after cessation of rotation, producing reverse stress in endolymph, sensation of reversed motion, and movement of objects to opposite side. Accompanied by after-nystagmus, heteronymous (L.) corresponding to sensation of subjective and objective motion. Compensatory head turning and body motion, as before, opposed to direction of nystagmus, are (R.) to restore balance, supposedly disturbed by rotation to left. This after-nystagmus and after-vertigo are easily observed after a few rotations when the subject attempts to stand and walk straight forward. It is also frequently seen in every day life, after whirling in dancing or skating. The dizziness does not come on until the rotation has ceased, but then takes place immediately, and the subject reels in the direction of previous rotation, after coming to a stand still, as if by some inward momentum. In fact, it is due to an attempt to correct the supposed disturbance of equilibrium or position. The vertigo may be inhibited, partially or wholly, by forced fixation away from the direction of motion, real or apparent. b — also shows the effect on the R. ampulla of turning to L. during passive or active rotation. The endolymph motion (serpentine arrow) is away from the R. ampulla, hence weak, and may be described as producing a weak sensation, of turning away from the right, and corresponding in every particular excepting intensity to the sensation reactions on right rotation in the left external ampulla. It is conceivable that after loss of function in the left labyrinth, this ordinarily subordinate sensation might be sufficient to cause an independent reaction and gradually compensate, in part, for the lost function of the contralateral labyrinth.

Nystagmus might be viewed as part of the somatic reaction having for its object the establishment of an optimum position of the eyes, corresponding with macular fixation of an external object coming into the field of vision from the source of apparent motion, or with corresponding subjective change of position; in other words, an equalization of visual stimuli, and as an aid to balance to be gained from vision by fastening on, and as it were being supported by a sense impression based on objective data. The nystagmus observed in disease and in experiments must be considered as running in habitual channels. The purposive element, of course, no longer appears, but that element can be detected and is logically to be explained. Thus there is no actual attempt at fixation in labyrinthine disease or rotational nystagmus, as is shown by persistence of this ocular motion when the eyes are closed or exclusion glasses worn, but the memory of this tendency, the local sign and tradition of this intuitive reaction persists and comes out, nonpurposively as a symptom.

TECHNIC OF CALORIC TEST.

To produce an even stream of long duration and what is important in labyrinthine suppuration, as little mechanical irritation through pressure of the stream as possible, it is not advisable to use a syringe. A large Politzer bag (Boenninghaus) or a fountain syringe hung a little higher than the ear may be used. A fine ear irrigating tip or an elastic middle ear catheter, introduced thorough an aural speculum is then carried close to the drum, an assistant managing the Politzer bag while the surgeon observes the eye-motions. These generally appear in from three-fourths to one and three-fourths minutes with intact drum, but in case of perforation they may be seen with good illumination in a few seconds, and last for several minutes. Cold water, at a temperature of about 50° to 60°, i. e., 40° lower than body temperature, is best for practical reasons as a like contrast could not safely be produced with warm water as the temperature would have to be over 130°.

Galvanic Nystagmus.—Breuer showed (1888, 1903) that on galvanic irritation of the exposed semicircular canal of the dove, head and eye movements took place, and that this reaction could be prevented by cocainization of the corresponding ampulla. After bilateral destruction of the labyrinth there is no reaction to galvanism. Similar conditions were noted in deaf mutes with total absence of labyrinth (Pollak, 1893). These observations exclude the possibility of cerebellar involvement in the galvanic reaction. The cathode acts like hot water, i. e., as an irritation corresponding to rotation toward the tested side. The nystagmus, as before, is toward the same side, and head motion to the opposite side. This appears to show that the galvanic current, running as it does from cathode, negative pole, to anode, positive pole, acts on the ampullar nerve terminals exactly as an endolymph current would when moving in the same direction, so that, translated into terms of subjective sensation, the passing of a current through, say the right horizontal canal from right to left would be felt as an endolymph motion would, i. e., as a turning motion to the right or irritated side. The anode causes the opposite reaction and is analogous to that of cold water, nystagmus to the "sound" side, and head turning to the side of irritation. Barany places the anode on the ear to be tested, and the cathode in the opposite hand. This is preferable to the transverse current through the head, as it allows either labyrinth to be tested separately. Nystagmus occurs more easily when the gaze is directed toward the cathode, i. e., again toward the source of apparent motion and of actual irritation, and toward the direction of subjective rotation. It can be elicited by currents of 2-6 milliamperes; while with the gaze in the opposite direction, 10-15 are usually required. Barany has noted that in labyrinthine destruction it is impossible to elicit galvanic reaction from the destroyed side, a complete analogy with caloric nystagmus. Babinski (1901) attached more importance to the compensatory head turning, which he found always took place toward the diseased side, when this side was stimulated either from cathode or anode. This "ear phenomenon" of Baginski is only found in unilateral disease of the static apparatus, according to Mann (1907); a significant observation requiring further study. Deaf mutes without rotational vertigo also show a loss of the galvanic reaction (Pollak, 1893).

GENERAL CONSIDERATIONS IN REGARD TO TESTS.

This general review of the history of investigation and the sketch of our present knowledge of the structure and functions of the inner ear must be supplemented, if it is to be applied to the practical needs of clinical otology, by a study of the functional reactions in health and disease. It is evident that even the most minute and elaborate tests of patients presenting symptoms of labyrinthine involvement can result in accurate knowledge of the underlying morbid conditions and lead to a definite diagnosis only if we have a fairly constant and definite basis of normal reactions from which to measure given variations. It may be said here, by way of parenthesis, that there still remains a great deal to be done in this field. A large number of carefully conducted tests on normal individuals is needed to show the reactions to rotation, caloric, galvanic, and postural irritation, the physiologic limits of balance, the determining factors of vertigo, and so on. Even then the subjective quality of many of the reactions, for instance vertigo, dizziness, loss of equilibrium, will tend to vitiate accurate conclusions by the interjection of the personal equation of both patient and observer. The elaboration of quantitative tests will of course be the ideal in this direction, and the reduction of all observations to a uniform basis will simplify the entire subject. As to the application of these tests in disease, there are of course, other points, pro and con, to be considered. Many of the tests, such as those of rotation vertigo and of balance angle, require special apparatus, a trained observer, much time, and more patience, besides the good will and co-operation of the subject. It is more than doubtful whether the tiresome and occasionally disagreeable rotation tests could be applied in private practice or even to any great number of clinic patients. The possibility of aggravating symptoms already present or of fancied injury on the part of the patient must be borne in mind. Galvanic irritation should be applied with the smallest perceptible current strengths, caloric nystagmus should be elicited without any mechanical violence, and the possibility of sudden falling should be thought of at all times, not only for the reasons just mentioned, but because, as we shall see, all these methods produce reactions in normal individuals and the results of our tests would be anything but instructive if they were conducted so hastily and roughly as to produce results which could be due to wide-spread irritation or the coincident reaction of distant sensory or central organs. This will be considered more in detail in the description of the clinical examination and tests of the labyrinthine functions.

THE REACTION OF THE NORMAL LABYRINTH TO FUNCTIONAL STIMULI AND EXPERIMENTAL IRRITATION.

Barany's tests were on normal subjects, patients with cranial injury, and on twenty-four cases of labyrinthine unilateral destruction. Up to the 50th year, age has no influence on the duration of consecutive nystagmus after prolonged rotation (10 turns). In normal subjects the average duration is 40 seconds, but there are marked individual variations. In dancers who do not "reverse," and are accustomed to dancing only to the right, the nystagmus is decidedly less marked after rotation to the right. Those who dance equally well to the right and left react like normal subjects. In health as well as disease after-nystagmus is more marked and lasting after rotation to the left. Repeated rotation tests show no signs of exhaustion in the reaction to turning; active fixation while turning tends to inhibit nystagmus. It is, accordingly, more marked if dark glasses are worn during the test, and lasts two or three times as long. Increased rapidity of turning lengthens the period of nystagmus and causes larger and more frequent excursions. The number of rotations up to 10 increases the duration of after-nystagmus, but with continued rotation (Barany has gone as high as 60), the latter again becomes less. After 20 or more rotations the after-nystagmus is followed by reversal (tertiary nystagmus) in the original direction of rotation, which is of central origin.* In many normal persons one or two turns are sufficient to cause after-nystagmus, if fixation-correction has been excluded by dark glasses. In case of destruction of the labyrinth of one side the nystagmus is always more marked and lasting toward the sound side. Smokers and neurasthenic subjects show increased irritability to rotation stimuli.

Ocular torsion or wheel motion of the eyes, first noted by John Hunter in 1786, which compensates for head tilting and persists until the vertical position is again assumed, must also be considered as a labyrinthine reflex for all but small angles of inclination, as noted previously. After destruction of both labyrinths in animals, this torsion does not take place, while it is markedly diminished, according to Barany, in deaf mutes and bilateral destruction of the labyrinth in normal beings. In unilateral destruction there is no alteration, but in unilateral irritation there is decided increase of compensatory ocular torsion. In normal subjects a tilting of the head of 60° corresponds to ocular torsion of 4° to 16°.

TESTS OF EQUILIBRIUM.—V. STEIN, 1895.

v. Stein distinguishes between static and dynamic tests, in accordance with the theory of Breuer and Mach. For the former he uses a movable inclined plane on which the patient stands. The angle of inclination is then increased until balance can no longer be preserved. Tests with this goniometer show that in the upright position normal subjects can be titlted backward 36 to 39°, forwards 26 to 30°, and tipped sideways 36 to 38° before they fall. When the eyes are closed the angle of fall is 3 to 4° less.

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*This "tertiary" nystagmus appears in the model as a gradual advance of the pointer to the vertical, from reverse position (secondary nystagmus) as the fluid comes to complete rest after rotation. It can be explained quite as simply as primary nystagmus by the local conditions in the semicircular canals, without recourse to the central organs for a basis of explanation.

Dynamic tests note the variations from a straight line or the tendency to fall in a certain direction when moving in various ways. The difficulty of preservation of balance and direction increases as we proceed from standing to walking and hopping, from the flat foot position to tip toe, from a support on both legs to a single point d'appui. The difficulties are still further increased by closing the eyes and by moving backward. By these progressive tests v. Stein and others believe it possible to detect slight disturbances of equilibrium which would otherwise escape observation, especially when the patients themselves are not conscious of them.

Normal subjects can stand with closed or open eyes, on both feet, for many minutes without marked swaying. Walking backward and forward, under like conditions, generally follows a straight line. In labyrinthine affections Romberg's phenomenon of swaying, and characteristic deviations from the straight line are frequently noted. The more difficult tests are not equally well passed by normal individuals. Practice and special skill account for the various reactions, and as Boenninghaus has observed, we may be in doubt in a particular case what we are to ascribe to individuality and what to disease. Yet we may assume that in simple clumsiness or lack of practice, the defect of static and dynamic balance will be the same on both sides, while in labyrinthine affections there is usually marked inequality. The following phenomena have been found characteristic, but not invariably present, in labyrinthine disease:

1. The affected side is the weaker. In standing or sitting the body inclines or falls to the diseased side. In walking or hopping there is a deviation to the diseased side, the patient does not stand securely on the leg of the diseased side, and the body can be more easily pushed over to the diseased side by the observer (Adler, 1894; Hinsberg, 1901; Krotoschiner, 1906).

2. The body muscles tire rapidly, so that in hopping, the jumps become shorter and shorter, until finally they "mark time", and are made on one spot (labyrinth jumps of v. Stein). (Fig. 9.)

3. Falling can be prevented by merely giving the patient a finger (Mann, 1907), as the slightest tactile sensations will compensate for the labyrinthine defect.

4. Falling is sudden, unexpected, and complete. The patient falls "like a log", without regard for possible injury and without the power of prevention or compensation, so that the necessary precautions should always be taken by those who institute these tests.

Finally it may be noted that tests with the dynamometer, the ergograph, and other instruments of precision show a decrease of muscular energy, tone, and muscle sense, and characteristic curves of muscular action, in labyrinthine disease corresponding closely to the results of animal experimentation.

NON-ACOUSTIC SYMPTOMS OF LABYRINTHINE DISEASE.

According to Barany, the diseases of the semicircular canals may be divided into three groups:

1. *Circumscribed labyrinthitis.* O. I. circumscript.—Symptoms due to irritation by hyperemia or toxic edema. Spontaneous nystagmus at intervals, elicited by head motion toward diseased side. Caloric test positive. Many diseases show this reaction. The most important are circumscribed labyrinthine suppuration, and fistula. Fistula symptom (nystagmus on air compression) may be elicited. Subjective sensations usually present. Marked vertigo, nausea, and occasionally vomiting. Partial deafness. *Rotation* toward diseased side increases spontaneous nystagmus, or brings it on after one or two turns. After-nystagmus to sound side slight. *Vertigo* present. *Disturbance of equilibrium.* Head turns away from diseased side, and with head upright, patient falls away from diseased side. (Attempted compensation.) If head is turned toward diseased side, patient falls backward, and vice versa. *Tonus* increased on diseased side. *Rotation* toward diseased side causes more marked and lasting somatic reaction, which may occur after one or two turns. *Recovery* with normally irritable static apparatus.

II. *Diffuse labyrinthitis.* O. I. P. A.—Loss of function due to *acute* and widespread suppuration, and indicated by the loss of caloric reaction. Total deafness. Spontaneous nystagmus to sound side, due to complete loss of function on diseased side and comparative excitation of remaining normal labyrinth is continuous, present in all positions, and tends to disappear. Tinnitus and spontaneous nystagmus occasionally persist for some time. Rotation toward diseased side causes slight somatic reaction, if any. Vertigo may be absent. Facial paralysis frequent. *Disturbance of equilibrium.* Tendency to fall or walk toward diseased side. Gradually corrected and compensated for by tactile and visual sense. *Tonus* markedly increased on diseased side. *Recovery* with unilateral loss of static function and loss of reaction to rotation.

III. *Chronic diffuse labyrinthitis.* O. I. P. C.—Loss of function due to old suppuration. Spontaneous nystagmus to sound side no longer present. Cerebral compensation and adjustment of temporarily disordered balance of labyrinthine stimuli. Caloric nystagmus absent. This may occur in cases which have progressed slowly from the start, with a subchronic course, nystagmus and vertigo never having been noted, and in cases of long standing labyrinthine suppuration with widespread destruction. Fistula symptom absent. *Rotation.* In II. and III. there is marked after-nystagmus to sound side. *Vertigo* absent. *Disturbance of equilbrium* shows gradual compensation. *Tonus* gradually restored, but muscular weakness remains.

SENSE OF BALANCE.

The sense of equilibrium and power of balance in human beings is a variable factor. Probably not congenital, as shown by the unstable equilibrium of small children, even when muscular power is great, and by the slight tendency to vertigo, which is rarely noted before the eighth year. Children are not subject to sea-sickness, can swing without becoming dizzy, or whirl in dancing or at play much more freely than adults. There are probably differences in the two sexes and at different ages. Neurasthenic or hysterical individuals are particularly susceptible to vertigo and nausaa. Occupation and training are of importance, and in tests of patients with labyrinthine disturbance it is well to ascertain what movements could formerly be performed correctly. There is a tendency for normal individuals to circle to the right in walking, and turning to the right especially in dancing is comparatively easy for the right-handed. Reversing is difficult for many, and this faculty should be inquired into. The tactile sense, muscle sense, tonus and sensations in the viscera combine to form one large uniform source of sensations as to our position in space. Visual sensations, sensu strictiori, and sensations of ocular muscle innervation, form a second. The third, labyrinthine sensations or disturbances, are generally subconscious and for that reason externalized and interpreted by the sensorium as sensations of objective or subjective motion. Normally, all changes of position or of equilibrium are associated with a sense of change in the field of vision, of the position of external objects as to us, an external disequilibration as opposed to the internal. Vertigo is produced when there is contrary innervation or discrepancy between the two sensation states. Thus even rapid and irregular motion of objects in the visual field will of themselves not cause vertigo, whereas very slight displacements will suffice when there is an absence of the usual corresponding sensations in the ocular muscles or those of the body.

OBJECTIONS TO BREUER-MACH THEORY AND V. STEIN'S TESTS.

Objections to conclusion from animal experiment, particularly section of the semicircular canals, as well as to deductions from the phenomena of their disease, in so far as it was assumed that these symptoms were evidence of local changes, were made from the beginning of investigations in this field. It was repeatedly claimed that the results were vitiated and the facts obscured by complicating disease or accidental injury of contiguous cerebral structures. Thus Hitzig (1871) believed that the phenomena of galvanic irritation nystagmus were due solely to cerebral irritation. Disproved by cocainization of the ampulla, and by observations on deaf-mutes. Boettcher (1875) laid stress on possible injury by traction on the nerve trunks in semicircular canal experiments. Weber-Liel (1880) considered brain irritation through changes of pressure in the aquaeductus cochleae sufficient explanation. v. Bergmann, in the same year, attributed Meniere's complex in the case of a fissure through the labyrinth to coincident traumatism of the brain. Lucae and Baginsky, judging from the effects of the air-douche and of injection of fluids into the middle ear, claimed that transmission of external pressure to the cerebrospinal fluid and consecutive central irritation or injury were the principal factor in disturbance of equilibrium. Baginsky in fact claimed that Meniere's complex was invariably dependent on cerebral lesions. Steiner (1888) claimed that all observed disturbances of motility could be explained by traction on the trunk of the auditory nerve, and that the phenomena were forced movements, not balancecompensations. Other investigators claimed that the varied data of animal experimentation were due to faulty observation, and that with careful section of the semicircular canals there are no disturbances of equilibrium whatever. (Boettcher, Lussana, 1872.) There may be extensive disease of the labyrinth without a trace of disturbance of equilibrium (Lucae, 1882). Kiesselbach and Steiner experimenting on fishes and lizards, were unable to find any disturbances after injuring the semicircular canals. J. D. Richards (Treatment of Purulent Affection of the Labyrinth, ANNALS OF OTOLOGY, etc., Sept., 1907) claims that sound waves affect the non-acoustic as well as the acoustic labyrinth, and gives, as a practical example, a case in which after exenteration of the entire labyrinth, loud musical sounds produced intense dizziness. This merely proves again what was well known, that the central trunk of the eighth nerve transmits acoustic as well as equilibrational stimuli, giving rise to cerebellar sensory reactions, and does not in any way invalidate the theory of the non-acoustic function of the labyrinth. Other objections raised by Richards on the basis of a case in which disturbed equilibrium, vertigo, vomiting and nystagmus were relieved by removal of semicircular canals, vestibule, and lower half of the first cochlear whorl, are due to his having confounded evidence of irritation with loss of function. The removal of a source of irritation and of false localization would naturally be followed by disappearance of double or faulty sensation. Richards recurs to the old theory of concomitant injury of central organs or of centripetal nerves which he resurrects under the name of "peripheral irritation," to which the interior of the labyrinth is supposed to be subjected in removal of parts of the labyrinth. His deduction that nystagmus and other reactions are not entirely due to the loss of parts of the labyrinth, because under anesthesia mechanical irritation of the interior of the vestibule or on the ampullary areas may produce nystagmus, is faulty, inasmuch as anesthesia does not in any way exclude the function of the organ of equilibration. Against the practical application of v. Stein's tests, Richards states they are subject to numerous errors, as, irregularity in length of limb, a badly arched foot, fear of falling, and claims that a patient if he be right-sided (handed) attempting to jump in a straight line, will deviate to the left and eventually tend so to fall if the eyes are closed. In the hopping test, the patient is not in contact with the floor long enough to gain a correct subjective appreciation through muscle sense of his proper relation to the objects about him. The disconcerting of muscular sense and suppression of orientation inherent in v. Stein's tests, create inequilibrium, so that if disturbed balance appears during the test we are at a loss to know what factor is responsible; in other words, it is not a differentiating test. In answer to these objections it can merely be said that they are not borne out by the results of practical experience. Normal individuals, whether flat footed, right or left handed, or otherwise asymmetrical, do preserve normal balance and walk straight forward or backward without difficulty, whereas, as shown by numerous observers, and again recently by Eagleton, labyrinthine disease does produce characteristic disturbances. No disequilibration is produced by v. Stein's test except the pathognomonic disturbance which we aim to elicit, and as to the tactile sensation during hops, if the length of time between them were insufficinet for orientation, we should all lose our balance when hopping, and there would be no tendency to fall to one side in preference to another. Richards' objections to theory and practice of equilibration and nystagmus tests are not well founded.

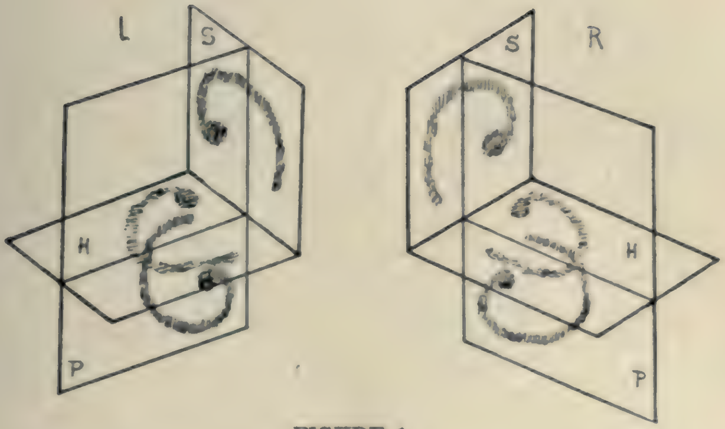

FIGURE 1.

Diagram showing relations of the semicircular canals (after Ewald). S, S, plane of superior or anterior; H, H, of external, horizontal; P, P, of posterior semicircular canals, seen from behind.

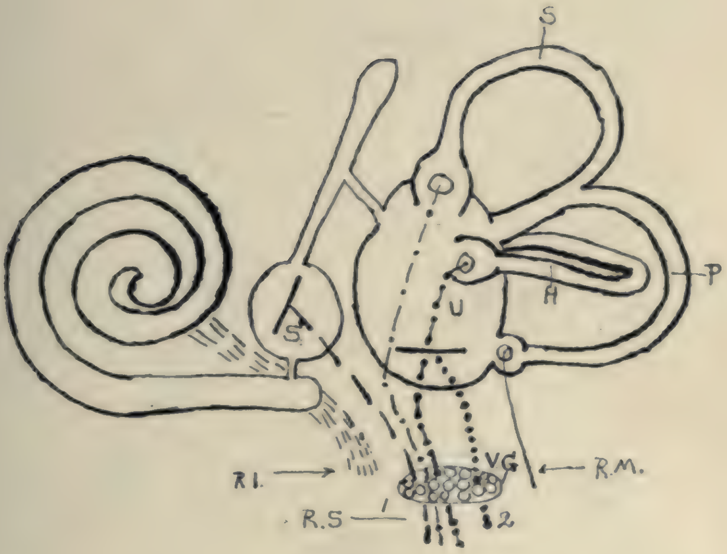

FIGURE 2.

Membranous labyrinth (left) from outer side. S, superior; P, posterior; H, horizontal semicircular canal; S^1, sacculus; U, utriculus with vertical and horizontal macula, respectively; R, I, ramus inferior (cochlear branch) of VIIIth nerve; R, M, ramus medius, from posterior ampulla; R, S, ramus superior; 1. first branch from macula sacculi, superior, and external ampulla; 2. second branch, utricular, from macula utriculi; V, G, vestibular ganglion.

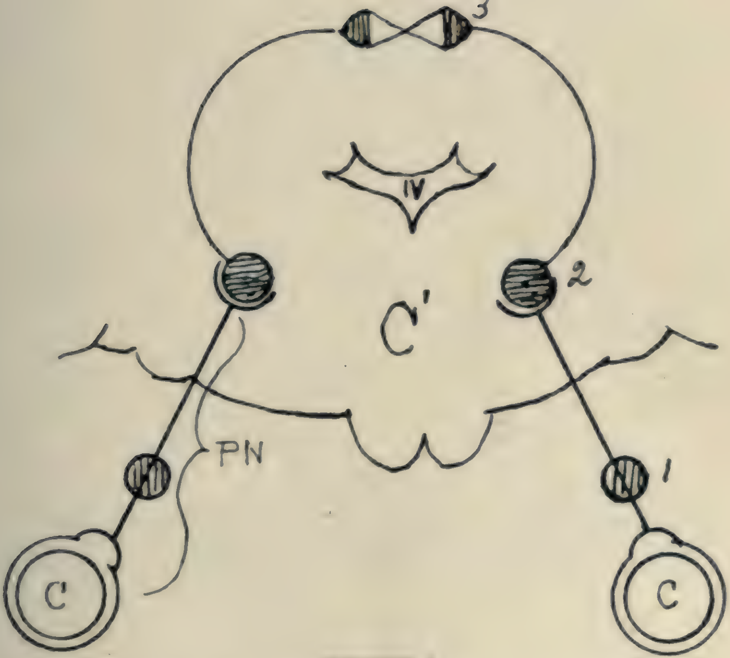

FIGURE 3.

Central course of vestibular nerve (after Boenninghaus). Diagrammatic cross section through pons and cerebellum. C, C, semicircular canal (vestibular apparatus); 1. vestibular ganglion; 2. dorsal nucleus of VIIIth nerve; 3. nucleus tegmenti; P, N, peripheral neuron; C^1, cerebellum; IV, fourth ventricle.

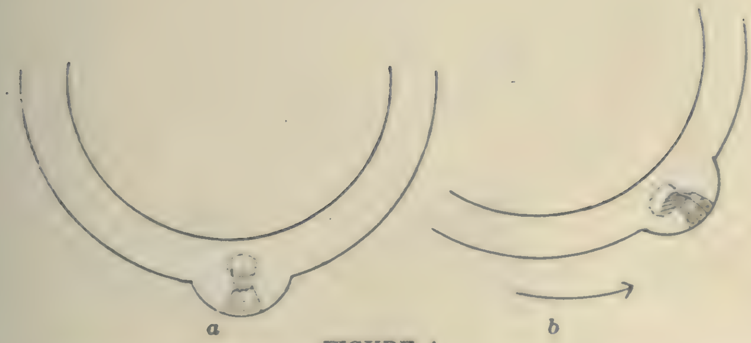

FIGURE 4.

Diagram of the action of inertia on sensory terminals in ampullae. a, canal and ampulla at rest; b, in rotation. In the latter the hairs of the sensory epithelium are dragged backward and irritated by the lagging of the endolymph.

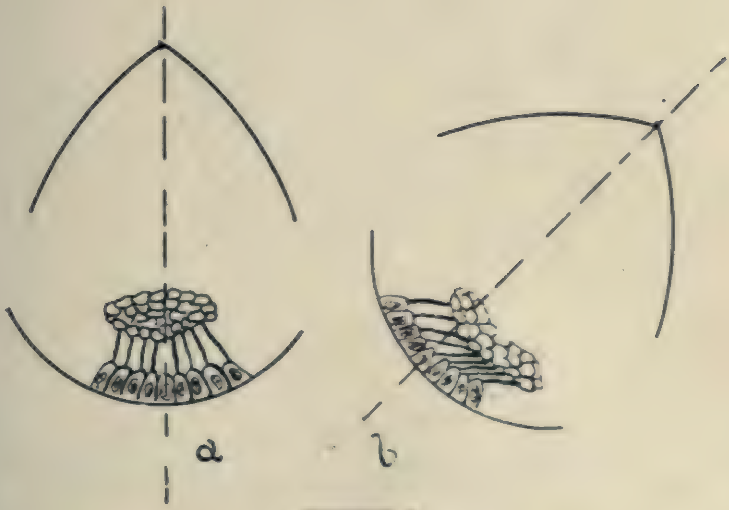

FIGURE 5.

Diagram of the action of gravitation on the sensory terminals of the vestibular organs (sacculus and utriculus). a, Vestibular organs at rest; b, inclined. In latter position the otolith plate causes by its weight and dragging, a distortion and irritation of the sensory hairs on the macula.

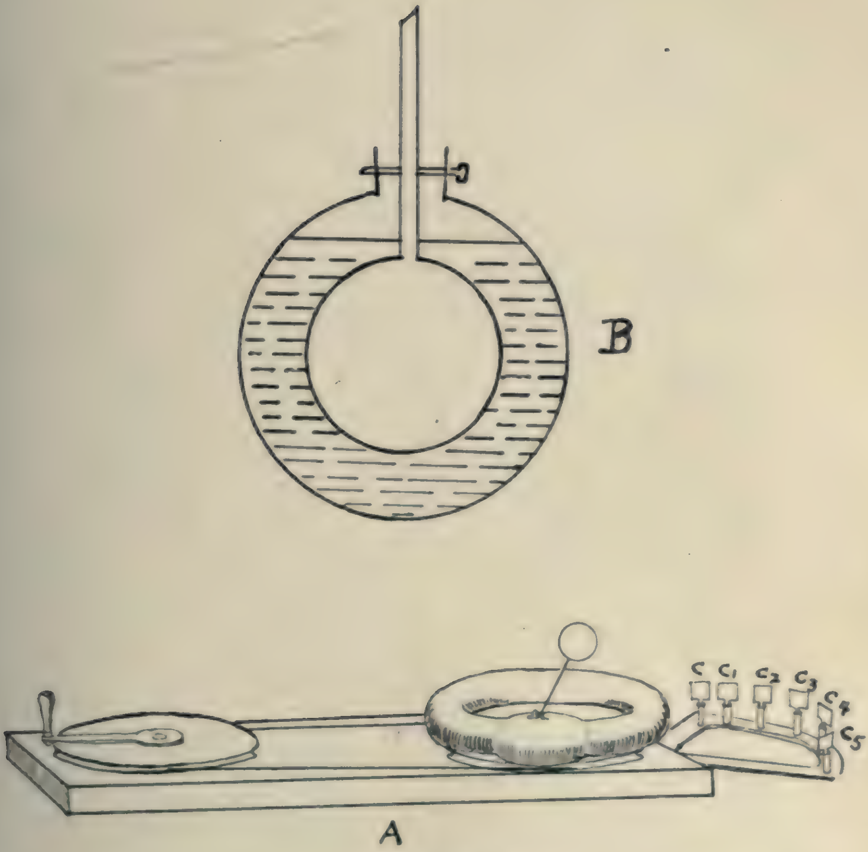

FIGURE 6.

Model showing mechanical principle of function of semicircular canals. A, Turn table. At right, a hollow ring of ¾ inch rubber tubing, nearly filled with glycerin and water, in which a small pendulum is suspended. B, Cross section through tube. Detail of pendulum. Beyond the table there projects a tin quadrant to demonstrate nystagmus. C-C^5, pegs covered with rubber tubing in which cardboard discs have been stuck to indicate external objects passing in rotation. On turning the disc, the fluid in the tube lags behind, at first, and holds back the pendulum, causing the indicator to tilt forward sharply in the direction of rotation. As the motion becomes more uniform, the indicator tends to stand upright. On cessation of motion the indicator reverses sharply, showing the momentum of the fluid in the tube, and the physiologic basis of sensation of reversed rotation. Acceleration or retardation of the disc is shown at once by an inclination of the indicator in the direction of turning.

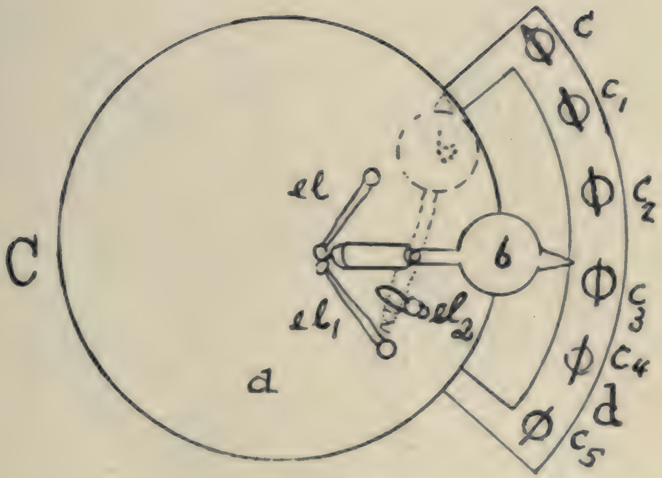

FIGURE 7.

Model showing mechanism of rotational nystagmus. C, Rotating disc: d, quadrant attached to base; C-C^5, pegs representing external objects, in which pointer, b, representing visual axis, engages; el, el^1, elastic bands to bring visual axis back to normal; el^2, attachment for forced abduction, as shown at b with dotted line. On revolving disc, representing motion of external objects while subject is turning, the pointer is held and caught a moment by each object in turn, springing back with a rapid recovery movement to normal position. On forced abduction with line of sight at a sharp angle with line of objects, the pointer is held much longer, the nystagmus, accordingly, appears more marked, when looking toward source of motion, and tends to disappear when pointer is in opposite position.

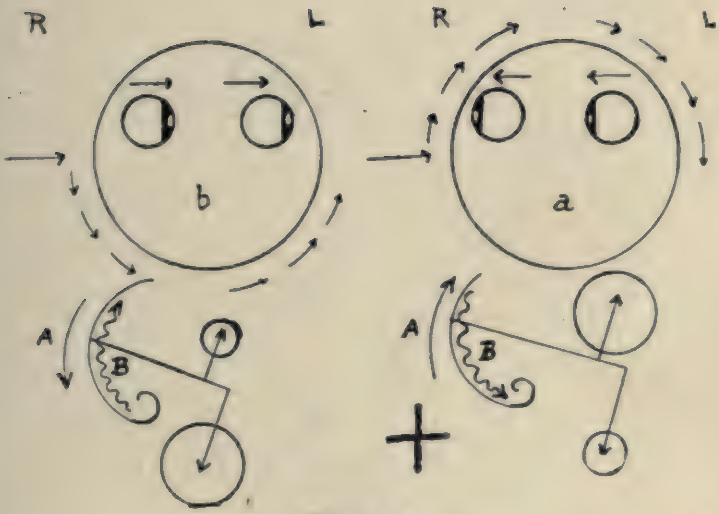

FIGURE 8.

Diagram showing direction of nystagmus, head-turning, and subjective sensation of motion, caused by caloric, mechanical, or galvanic stimulation. a, b, Face seen from in front, indicating direction in which eyes are turned, nystagmus; a, positive stimulation,-|- as by syringing right ear with hot water, turning patient to right, compressing air in right auditory meatus, or closing galvanic current with cathode on right ear. Nystagmus to R (small circle with enclosed arrow), head turning to opposite side; L (large circle with enclosed arrow). Movement of endolymph (serpentine arrow at B) strong, toward ampulla, away from source of irritation and from direction of real or sensory rotation, toward opposite side, giving subjective sensation (long curved arrow at A) of rotation to R, irritated side. b, Negative,—stimulation, as by cold water syringing of right ear, rotation to left, rarefaction of air in right meatus, anode closure on right ear. Signs as before.

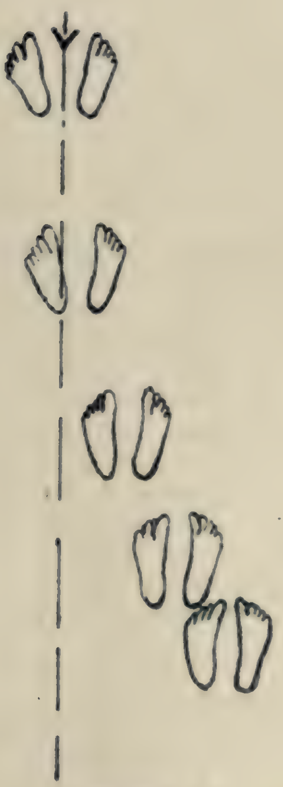

FIGURE 9.

"Labyrinth Hops," v. Stein. Hopping backward, in direction of arrow, in disease of right labyrinth.

XXVIII.

CHRONIC RECURRENT SUPPURATIVE OTITIS MEDIA AND ITS RELATION TO MASTOID AND INTRACRANIAL COMPLICATIONS.*

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INTERMITTENT OTIC DISCHARGE MORE DANGEROUS THAN THE CONSTANT VARIETY.

In considering the subject of chronic recurrent suppurative otitis media, I will endeavor to demonstrate that this particular form of aural disease is relatively productive of more complications, intracranial and otherwise, than the continuous variety. I have been particularly impressed with the importance of this subject from the fact that the belief still persists, both among the profession and the laity, that an aural discharge which is intermittent is of little importance, the assumption being, therefore, that the underlying disease is never dangerous to life. This fact clearly illustrates that the pathology and prognosis of these conditions are not generally understood, notwithstanding the marked progress which otology has made in this direction during the past decade.

OUR DUTY TO THE PROFESSION AND THE LAITY.

For years past many of us have been persistent and untiring in our efforts to impress upon the profession the importance of aural disease, and to indicate the obligation due our patients in the prompt recognition and treatment of the same. Like all weighty medical problems, the evolution has been slow, but we have at least established a fairly good general conception of the acute diseases, their dangerous complications and oftimes tragic sequelae. As the general surgeon has persistently urged, year after year, the necessity of early operative interference in appendicitis, so must we labor to secure a proper realization of the actual dangers of a discharging ear.

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*Read before the American Laryngological, Rhinological and Otological Society, at Pittsburg, Pa., May 29, 1908.

Furthermore, we must not only recognize this condition, but must properly appreciate its gravity and demand the adoption of prompt and efficient measures for its relief.

This widespread indifference to a very grave problem is the natural outcome of continued apathy on the part of the medical profession, and if the Fellows of this Society are to conserve her honorable traditions, it becomes the immediate and imperative duty of each and every one of us to spare no effort, however self-sacrificing, to place before the profession some plain facts relative to the ever-present dangers of an otorrhea. We must, therefore, accept the obligations and responsibility created by such circumstances, and resolve to diffuse, in the most effectual manner at our command, such information as will throw proper and responsive light on this subject before the great body of practicing physicians, who will in turn be moved to action and will thereby become the legitimate instructors of their patients, the laity.

VALUE OF PROPHYLAXIS.

In this connection I wish again to take the opportunity of pointing out the great value of prophylaxis as applied to mastoid and intracranial involvement, complicating acute tympanic disease. In the primary stage we find a simple hyperemia and swelling of the mucosa, together with some seromucous exudate. At this particular period probably four-fifths of all cases will yield promptly if the well-known simple measures for their relief are immediately applied, whereas the vast majority of all serious complications are directly traceable to the unpleasant fact that these primary measures have been partially or wholly neglected.

I am not unmindful of certain cases complicating the exanthemata, influenza, etc., which seem to be virulent and purulent from their very inception, but even in such cases a free incision of the membrana tympani before bulging occurs will usually prevent serious involvement of the accessory cavities and adjacent structures. Such clearly defined facts should, therefore, convince us that a discharging ear, whether of the acute or chronic form, is frequently the result of woeful ignorance or indifference, and that the subsequent mastoid and intracranial implications are mainly chargeable to the same cause.

At times we are confronted with the statement that an otorrhea has existed for many years without producing untoward symptoms. So long, therefore, as the patient experiences no actual suffering he is content, often by the advice of his physician, "to let well enough alone." This viewpoint is quite natural and excusable on the part of the patient, but it does seem inconceivable, in this day of progressive and preventive medicine, that those charged with and professing to conserve the public health, should so far forget their obligations or misinterpret their humane duty.

As a body, those practicing the learned profession of medicine are among the most honorable, and certainly the most self-sacrificing men to be found in any calling; consequently, this lackadaisical attitude toward aural disease that we too often meet with can only be attributed to misconception or misinformation as to the worth and importance of the subject. Such being the case, I again wish to proclaim this society, individually and collectively, the disseminators of such information or instruction as may be necessary for the betterment of our profession, which, when viewed in the light of civic duty, must be accepted as an imperative obligation due to our fellowman.

ILLUSTRATIVE CASES.

In order to properly illustrate the destruction that frequently accompanies a recurrent suppurative otitis media. I will briefly relate the histories of three typical cases, which I have recently operated upon.

CASE 1. *History.*—Male, T. W. D., aged 44 years. There is some doubt as to whether he suffered from aural disease early in life. During the past three years he has had a recurrent suppurative otitis media of the left ear, the primary cause of which is unknown.

The patient came under my care at the Jefferson Medical College Hospital, April 3, 1908, stating that for the past three or four days he had suffered very severe pain, not only in the ear and over the mastoid process, but also on that entire side of the head, so much so that hypodermic injections of morphine were necessary to relieve his suffering. The history furthermore states that these acute exacerbations occurred about every three or four months. In the interim the ear would be entirely dry and free from pain. Prior to the last attack, however, he was free from all aural symptoms for about eight consecutive months.

The examination showed the external auditory canal to be filled with a copious, brownish-yellow, foul-smelling pus. There was a good-sized carious opening through the posterior wall of the external auditory canal, and communicating with the mastoid antrum, through which pus was escaping. There was much swelling, redness and tenderness over the mastoid process, extending upward to the superior border of the temporal bone, anteriorly to the outer canthus of the eye and posteriorly to the occipital protuberance. Pressure on this swelling would force a large quantity of pus through the carious opening already mentioned into the external auditory canal. The posterior half of the membrana tympani was destroyed; the anterior portion, however, remained intact and was quite normal in appearance.

Patient's temperature at this time registered 103°. Operation was advised and refused. The following day he returned, still suffering, the temperature rising to 104° at noon. Operative interference was strongly urged, but again refused. The next day he came again, at noon, his temperature at this time registering 105 1-5°. He was not able to secure sleep or relief from pain even under the influence of hypodermic medication. He now readily consented to an operation, and was admitted to the hospital for this purpose.

Operation.—A free incision through the soft parts evacuated a large quantity of pus. This incision was carried from the tip of the mastoid to about one inch above the superior border of the auricle, and then forward to the outer canthus of the eye for a distance of about two inches. A transverse incision on a level with the mastoid antrum was carried almost to the occipital protuberance. This large field of operation was necessary on account of the extensive osteomyelitis present. In this particular case the extension from the diploe, which is usually first involved in cases of osteomyelitis, was external rather than internal, probably taking the course of least resistance. The entire external plate of bone was very dark, in some spots almost black, with numerous points of hemic ooze. All this area of necrotic bone was removed. There was a large carious opening in the mastoid process a little below the level of the antrum. The remaining part of the process was also discolored and showed many bleeding points. The necrosis was so great that the whole process was removed, including the tip. The sinus, as so frequently occurs in these chronic cases, was situated rather superficially and far forward, so much so that part of the posterior auditory canal acted as the anterior bony wall of the sinus. The necrotic process had exposed the entire sinus, which was bathed in pus, and covered with a thick layer of what seemed to be healthy granulation tissue, with the exception of one part. The granulation tissue was removed on account of some bleeding, and this bleeding was increased by the removal of this tissue, although no opening in the sinus could be found. The dura was largely exposed, both through the tympanic roof and the antrum. The discoloration of the dura through the antral opening was so marked that it suggested the possibility of some intradural abscess formation. A hypodermic needle was introduced for the purpose of diagnosis, but with negative results.

The septic temperature ranged from below normal to 106°, and continued for some days after the operation. The man at times became quite delirious, and the whole picture resembled one of septic sinus thrombosis. My own conviction, and the judgment of the various consultants, favored the opinion that the man was suffering from a general septicemia. Hence, no further operative interference was resorted to. The wisdom of this conservative decision was borne out by the fact that after the patient had passed through superficial multiple abscess formations in various parts of his body, he made a complete recovery.

Blood cultures and differential blood counts were made at various stages of this patient's illness. The former demonstrated the presence of the staphylococcus pyogenes albus, and the latter showed, as might be expected, a marked hyperleukocytosis and a decided increase of the polynuclear percentage. This relative ratio between the hyperleukocytosis and the polynuclear percentage continued throughout his illness, clearly demonstrating the value of the blood count in all septic conditions.

Frequent examinations of the eye-ground gave negative results.

One point of interest and importance to which I especially wish to direct your attention, is the fact that we can hardly conceive that all the bone destruction shown in this case could have occurred in the three or four days which marked the acute exacerbation of the chronic recurrent suppurative otitis media, and yet the patient and his family claimed that for eight months prior to the date of this acute exacerbation he did not suffer the slightest inconvenience from his ear and that the same remained entirely dry during that period.

This brings up for consideration the extensive pathologic changes that apparently take place without causing the patient any discomfort. We are frequently surprised at the extensive necrosis met with in operating for the relief of mastoid disease. This remark applies both to the acute and the chronic form of mastoid disease, but of course is much more common in the latter. From this experience, especially during epidemics of influenza, I am convinced that bone destruction does sometimes occur with little or no inconvenience, until a certain point has been reached. In other words, this furnishes us an additional argument in favor of comparatively early operative interference.

The fact that this patient was able to attend the dispensary service on three consecutive days, with a temperature ranging from 103° to 105°, would indicate that he possessed an unusually good constitution, and that he could not have had any serious intracranial involvement.

CASE 2. *History.*—Another case which well illustrates the point under discussion is that of H. D., aged 18 years, who suffered from a recurrent suppurative otitis media of the right ear from early childhood, the trouble recurring at intervals ever since. At times the discharge would cease for a period of about one year, the membrana tympani completely regenerating in the interval, the pain, however, being much worse during the period of cessation of discharge. Whooping-cough is the only disease from which the patient ever suffered, and this is said not to have had any influence on the aural condition.

An acute exacerbation occurred about two years ago and was very severe, the discharge lasting until he applied for treatment at the Jefferson Medical College Hospital, October 11, 1907. Examination at that time revealed the presence of a slight discharge in the external auditory canal escaping from a small perforation in the anterio-posterior part of the membrana tympani, the remainder of the membrane being intact and looking fairly healthy. The Eustachian tube was patulous, an increased amount of secretion being forced through the perforation by the use of Valsalva's method of inflation. Deep pressure over the mastoid process elicited some tenderness.

In order to provide for better drainage, the membrana tympani was freely incised and the patient placed on the usual line of treatment.

November 23, 1907, or about five weeks later, the drumhead was found to have entirely regenerated and was quite normal in appearance, the patient, however, complaining of severe pain over the mastoid process and the right side of the head, although, with this exception, there was not the slightest evidence of the classical symptoms of mastoid disease, as evinced either by redness or swelling over the process or by any inflammatory condition of the drumhead. There was, however. some drooping and slight redness of the superior and posterior wall of the external auditory canal.

When the patient first applied for treatment, the mother was advised that an operation would probably be necessary. The condition had now improved to such an extent that we advised deferring operative interference. This did not meet with the approval of either the patient or his mother, the latter stating that the boy had been going through this process practically all his life, and she was convinced that the recurrence of pain on that side of his head would soon be followed by a reappearance of the discharge and that at such times he usually became quite ill, with high fever, severe headache, nausea, vomiting and great prostration.

Operation.—Heath's operation was performed on December 6, 1907. Here again the mastoid was found to be of the diploic variety, the accompanying osteomyelitis extending back almost to the occipital protuberance. This, of course, offered a ready explanation of the pain on pressure over the mastoid and posterior to the same. As in the former case, extensive necrosis was found, especially over the sinus, exposing the same for almost its entire extent.

The sutures were removed and the dressings changed six days after the operation. The wound was dressed every second day until January 2. 1908, when the patient was allowed to leave the hospital apparently well. On February 15, 1908, or about two months after the operation, the patient returned, the discharge having entirely ceased and the hearing having shown marked improvement. On April 17, or about four months after the operation, the patient again reported for examination. The membrana tympani was now entirely regenerated, and there was a complete cessation of the discharge.

The hearing had improved almost to normal, and he was practically free from all discomfort.

The result, in so far as the hearing is concerned, shows the importance of resorting to a Heath or a modified radical operation in all suitable cases.

The destructive nature of a recurrent suppurative otitis media, to which little or no attention is usually paid, is well illustrated by a patient from the far West on whom I recently operated.

CASE 3. *History.*—Married woman, aged 38 years. Without apparent cause she has had a recurrent suppurative otitis media of the left ear practically all her life. Up to eight years ago the discharge would appear about every two months. At that time, however, it ceased until nine months ago, when the acute exacerbation was ushered in with characteristic headache, nausea and great lassitude, extending over a period of several days, until the discharge again appeared, which in turn relieved the pain considerably, as so often occurs. The otorrhea, however, had continued during this entire nine months. In June, 1907, she had an acute suppurative otitis media of the right ear, which was the first time this ear had been involved.

The patient consulted me April 7, 1908. She was found to be suffering severely with headache, confined to the left side. There was no swelling over the mastoid process, but she suffered from rather severe pain on gentle pressure over the mastoid, and extending for some distance posteriorly. There was a copious discharge escaping from the external auditory canal, of a brownish-yellow character, streaked with blood and very offensive. The entire membrana tympani in this ear was practically destroyed, as well as the ossicles, which, however, were said to have been removed on a former occasion. The superior and posterior wall of the external auditory canal was very red, extremely sensitive, and drooping. The hearing was practically nil.

Operation.—In this patient we found the cortex exceedingly hard, it being flint-like in character. The sinus was pushed far forward and exposed by the necrotic involvement. There was some necrosis of the bony roof of the mastoid antrum, which, on being removed, evacuated considerable pus from an extradural abscess.

The radical operation was performed in this case. The patient made an uninterrupted but rather slow recovery, on account of suffering from an intercurrent attack of purpura hemorrhagica with hemophilia, the bleeding taking place at many points over the entire surface of her body, but more especially in the throat and gums. Although no undue hemorrhage was met with at the time of the operation, there has been a tendency since that time to have some bleeding from the mastoid cavity. On the other hand, up to the present time there has been no recurrence of the purulent secretion. The vertigo from which she suffered prior to the operation has continued at intervals. This may be accounted for by some possible hemorrhagic invasion of the labyrinth.

EXACERBATIONS CAUSED BY INFLUENZA.

Of seventeen mastoid operations performed by me in six consecutive days, during the second week of January, 1908, seven were of the recurrent variety. All showed extensive bone destruction, exposing either the sinus or dura or both. Two led to the evacuation of pus from a temporo-sphenoidal abscess, through the mastoid antrum route, and one assumed the Bezold's variety of mastoid disease. Previous duration of the disease ranged from twenty-seven months to thirty-one years. The acute exacerbations occurred in two cases about every two months, and in the others about every two years or more. Four of the patients had not noticed any discharge from the ear, nor experienced the slightest discomfort from the same for a period of three years or more. In one patient the ear had not discharged for six years when a sinus thrombosis developed. In all these patients the acute exacerbations were complications of influenza.

Of about one hundred mastoid operations recently performed, twenty-one were of the recurrent variety. Of this number, eleven showed unexpected necrotic exposure of the sinus or interior of the skull. Two died during the acute exacerbation, one from an old encysted temporo-sphenoidal abscess, which ruptured as the result of an injury to the head, and the other from an infectious meningitis. In this latter case free pus was found in the cavernous and petrosal sinuses, the lateral sinus being apparently uninvolved. This patient enjoyed good health until his final and fatal attack, his previous exacerbation occurring more than twelve years before. The primary ear disease developed from epidemic measles forty-two years prior to this date.

During an attack of suppurative otitis media, especially when long-continued, the mucosa lining the tympanic cavity and accessory cavities first undergoes an ulcerative process, and this in turn becomes macerated and peels off, leaving a greater or less area of exposed osseous surface. With the protective and nutritive coat removed, the bone becomes an easy prey to the destructive influence of pathogenic microorganisms Consequently it is hardly to be wondered at that so much necrosis of the bony structure should occur in such a large number of suppurative cases.

The location of the perforation in the membrana tympani would seem to offer some points in the diagnosis of serious intratympanic as well as mastoid caries. Although the rule is not constant, it nevertheless does occur with sufficient frequency to warrant the assumption that perforations situated superiorly and posteriorly in Shrapnell's membrane, and more especially if they exist in connection with drooping of the posterior and superior wall of the external auditory canal, are definite diagnostic indications of extensive caries of the tympanic cavity as well as of the mastoid process.

During suppuration the mucous membrane of the tympanic cavity shows hypertrophy, with the formation of vegetation or papillary growths, which may be large or small, discrete or confluent, extending along the inner wall of the cavity to the orifice of the Eustachian tube. As suppuration continues the epithelial cells desquamate and form large, whitish-yellow masses, which are composed of inspissated secretion and desquamated epithelium. These may be retained for an indefinite time unless infected by the staphylococcus, when they are converted into a foul-smelling discharge, consisting of bacteria and granular debris. Occasionally they assume a pearly white appearance and remain in the middle ear cavity as homogeneous masses or cholesteatomata.

The inward growth of the epidermis of the external meatus towards the tympanic cavity without the formation of cholesteatomata takes place more often than is generally supposed. On the other hand, secondary cholesteatomata are very often caused by the intrusion of the epidermis of the external meatus into the middle ear. They also develop in the middle ear itself, as shown by the presence of non-nucleated squamous cells. These cholesteatomatous masses may attain a considerable size without the least sign of caries or necrosis. On the other hand, it must be remembered that these epithelial accumulations or growths are probably more destructive, both to the soft and osseous structures, and cause more serious mastoid and intracranial complications than any form of infection.

In long-standing cases the horizontal walls of the semicircular canals may become eroded and the internal structures become the seat of inflammatory changes affecting the aqueductus vestibulae and the aqueductus cochleae. This in turn may cause a cerebellar abscess, resulting from suppuration within the labyrinth, the pus having followed the auditory nerve and burrowed its way through the internal auditory meatus.

More especially during epidemics of influenza we meet a large number of serious mastoid cases, the cause of which cannot be definitely explained. In other words, from the patient's history and in so far as can be seen, the middle ear was not involved. In cases of this kind, the bacteria and their toxins undoubtedly must have reached the mastoid by a hematogenous form of infection,

As is well known, recurrent cases may remain quiescent for some years, when the patient suddenly complains of severe pain in the head and ear of the same side. There is general hebetude, followed by chills, fever, vertigo and vomiting, symptoms which frequently usher in some intracranial involvement

In cases of a mild nature, or where an intracranial complication is neither present nor in the course of development, the intensity of the above symptoms will be more or less modified, as the severity of the attack is governed entirely by the underlying involvement.

DIAGNOSTIC VALUE OF LEUKOCYTOSIS.

In considering the bacterial invasion of the temporal bone and the adjacent structures, we often speak of the presence and value of a leukocytosis. In this connection it is well to remember that a leukocytosis is a perfectly normal condition in the healthy state, and is, in fact, nature's method of providing scavengers for the destruction of the numerous microorganisms that are constantly invading the human economy This leukocytosis, therefore, must necessarily vary in degree, depending entirely on the quantity and character of the toxins to be destroyed.

In the great majority of cases a normal leukocytosis is quite sufficient to destroy the hostile microbic incursion, but when the pathogenic organisms are numerous and virulent, a battle royal ensues between the leukocytes, which resent the hostile entrance into the possessions or domains of another, and the invading microbic army. In order to successfully meet and destroy the enemy, nature greatly increases the normal percentage of leukocytes, in which state the term hyperleukocytosis or leukopenia, which naturally signifies an entirely different condition. The normal number of leukocytes varies from 6,000 to 10,000 to the cubic millimeter. Hyperleukocytosis indicates an increase to 15,000 or more, and hypoleukocytosis or leukopenia is a decrease from the normal amount.

An examination of the blood is often an important link in the chain of symptoms which goes to make up the diagnosis in a septic process, but this examination must consist of the differential leukocyte count as distinguished from merely determining the presence of a hyperleukocytosis on the one hand, or leukopenia on the other.

The condition in sepsis, in the majority of cases, shows a decided increase in the leukocytes, but this is by no means constant, for as great an authority as Grawitz states that "not rarely active inflammatory processes with suppuration may be present without hyperleukocytosis occurring."

Leukocytosis gauges the combative powers an individual possesses and can use against the infection by which he is attacked, but it does not show the strength of that infection. Thus, in trifling infections in which the activity of the leukocytes is insufficient, and also in fatal cases where the system is overwhelmed by the toxins, hyperleukocytosis does not develop and even leukopenia may be absent. Facts such as these render the occurrence of hyperleukocytosis in sepsis an inconstant phenomenon. An early development and rapid increase in the number of leukocytes is decidedly in favor of septic disease. We have, however, a good index to the severity of the septic process in the relative percentage of polynuclear leukocytes. Normally there is present a variation of from 59 to 68 per cent of these cells, with a mean percentage of 61. Therefore, if a patient presents a blood picture in which the relative polynuclear percentage is above 80, it indicates absorption of pus from some part of the body. Pus is seldom found with a relative percentage of less than 80 unless the patient is a child, in which case it may be as low as 73. However, pus should be suspected and looked for in all cases in which the polynuclear percentage is above 70.

In addition to the hyperleukocytosis there is at times another element which may be determined from the examination of the blood, namely, the demonstration of the special microorganism, often in almost pure culture. It is never sufficient to content ourselves with a single culture of the blood, but this should be repeated, and in addition various culture media should be employed.

The relations of the ear to sepsis are extremely important. Ponfick examined the ears in one hundred children whose ages varied from one month to four years, and who had succumbed to various diseases. The autopsies revealed that 78 times there was bilateral, and 13 times unilateral, otitis media. In only nine cases was the middle ear normal.

From the foregoing it would seem that we have at our command a valuable asset, which, when properly worked out, may throw much invaluable light on certain forms of sepsis. For example, in cases of recurrent suppurative otitis media, during the stage of apparent inactivity, or in other words, between the periods that mark the acute exacerbations, it may be possible, by an examination of the blood, to demonstrate that a retrogressive metamorphosis is in actual progress. The value of such findings would at once become apparent if we were enabled thereby to appreciate such conditions, even in the absence of certain localizing symptoms.

XXIX.

SOME SURGICAL PRINCIPLES ESSENTIAL TO THE CURE OF FRONTAL SINUS EMPYEMAS.*

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A review of the recent literature of the surgical treatment of the pyogenic affections of the frontal sinus shows clearly that much difference of opinion exists among operators of experience concerning the proper plan of procedure. The varying degrees of severity of the disease that may be present in the sinus, and the several accessory conditions that may occur in a given case, often make the choice of plan debatable; but in those cases of suppuration of the sinus where the symptoms are quite similar in every essential, it would seem that too great a difference of opinion exists among operators of apparently equal prominence and experience. Such a difference of opinion is unfortunate for those operators of less experience, who as a result, too often feel a sense of doubt and uncertainty when contemplating the performance of frontal sinus surgery. The diversity of opinion emphasizes the fact that this class of surgery is often difficult, and that ideal surgical measures have not yet been established. To some extent, no doubt, the present views concerning frontal sinus surgery are the result of the rather rapid evolution of the rhinologist from a condition of practice in which his chief duty, only a few years ago, seems to have been to cleanse and medicate the mucous membranes of the upper air tract, to that which today may require the execution of the most skillful surgical measures, involving not only the nose, but also the more distant and intricate structures of the entire head. This evolution from the erstwhile rhinologist who was a sprayer of noses to the present rhinologist who must be a high type of surgeon has in some measure been responsible for two sets of operators, viz., those who believe that all, or nearly all, cases of accessory nasal sinus suppuration should be treated by the intranasal route, and those who hold that while the simple sinus empyemas may usually be thus successfully managed, when much actual pathologic change has taken place in the structure within the sinus or its bony environs, external surgery should usually be chosen.

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* Candidate's thesis, American Laryngological Association.

The discussion which continues between that class of surgeon who maintains, as does Ingals, that 95 per cent of all cases of suppurative frontal sinus disease can be cured by the intranasal route, and that class which believes that the greatest certainty of cure, and the least danger to the patient lies in the methods of operation by external surgery, has brought forth some good arguments in support of their respective beliefs. The intranasal surgeon points out in support of his position the alleged facts that ample drainage, and the free admission of air into the diseased sinus will cure all except the worst cases, and that as a result of the concealed operative measures required, no unsightly scar remains. The chief argument of those who prefer external surgery and the only one they consider absolutely necessary to make is, that thereby only can perfect drainage be secured, and the diseased structures be completely removed. The more or less scar tissue which results from external surgery of the sinus, while always an objectionable feature, is by this class of surgeons regarded as a sequence absolutely necessary to a successful issue, and therefore entirely justifiable.

This paper is not, however, greatly concerned in the dispute between the two classes of surgeons, for both admit that a certain number of all cases of suppurative sinus affection must be operated by the external route, and that a certain percentage is also curable by intranasal procedures; but it is rather with the surgical principles essential to the cure of sinus empyema which should govern the method which is chosen, that we shall be most interested.

Good results from the practice of any method must depend largely upon the proper application of certain well established surgical principles. Aside from an aseptic technic, which should be as perfect as possible in every case, the principles upon which all successful sinus work must depend are: 1, the complete removal of all pyogenic structures; 2, provision for ample drainage of the operated parts; 3, healing the diseased cavity either by a healthy, non-suppurative lining, or by the complete obliteration of the space by means of granulation tissue. The true value of either the external or intranasal method of treatment of suppurative frontal sinus disease can, therefore, be excellently judged by subjecting each plan to the standard of requirements of the above surgical principles. Of course in addition to these principles, the length of time the patient must suffer or be inconvenienced by the necessary treatment subsequent to each, and the actual danger to life resulting, although not surgical principles, are nevertheless often the result of the application of correct or faulty principles, and must, therefore, in justice be considered. Let us apply these principles first to the method of treatment by intranasal drainage.

The efforts of operators like Ingals, Halle, Good and others to cure frontal sinus suppuration by the intranasal route, and the ingenious devices which they have perfected for entering and draining the frontal sinusus, should receive the heartiest commendation. Undoubtedly a large percentage of cases in which structural changes have not been extensive, and in which the anatomic formation of the sinus is normal, can be and are cured by the simple provision thus made for good drainage. When, however, anomalies in the anatomic structure of the sinus are present, and when extensive pathologic tissues have been formed within, drainage alone is not often sufficient to bring about a cure; for in most instances it is impossible to remove this diseased pathologic content of the sinus, or to correct its anomalous structure through the comparatively small opening which it is possible by this method to provide for the drainage. Moreover, in case of the frontal sinus it has been considered a settled point that it is not possible or safe to enter the sinus with instruments intended for the enlargement of the infundibulum, in a considerable percentage of cases. Mosher (Candidate's thesis for American Laryngological Association), after a study of the applied anatomy of the frontal sinus in 100 wet specimens, and 50 cleansed skulls, states that it is only by chance that the nasofrontal duct can be catheterized. Turner (Accessory Sinuses of the Nose) says, the variation in the frontal sinuses which have been described, sufficiently indicate that the passage of the probe through the anterior nares toward the frontal sinus may meet with some opposition. Occasionally a narrow or tortuous duct, or the small size of the ostium frontale, may prevent the probe reaching the sinus. Beaman Douglass states concerning this point (*Laryngoscope,* May, 1904): "One is often mistaken in believing that he has entered the frontal sinus, when, as a matter of fact, his instrument is within an enlarged anterior ethmoidal cell (bulla frontalis). All who have extensively examined the frontal sinus either in the wet or dry state of the skull must have observed that in a considerable percentage of instances an anterior ethmoid cell projects itself into the frontal sinus either external or internal to the sinus orifice of the duct, and that the mouth of the duct lies partially under such cell. In this class of case catheterization of the frontal sinus, when successfully performed, must always result in the entrance of the instrument through the broken down ethmoidal cell wall, through which it has been forced. Douglass has, furthermore, pointed out the fact, which has also been observed by others, that the internal wall of the frontal sinus frequently lies so low above the orifice of the duct that any cutting instrument, even if successfully introduced through the nasofrontal duct, would immediately penetrate the cranial cavity and would most probably set up an infective meningitis. On this point Mosher states that if there is a sufficient basal relationship between the floor of the frontal sinus and the nose, a burr can be pushed up through the duct into the sinus, the burr running on a probe as a pilot. Concerning this plan of entering the sinus, Mosher observes that, on the cadaver, beautiful specimens can be made in this way, but that he is always afraid that the practice of the method will make cadavers of some of his patients. The literature of the subject, therefore, clearly indicates that few operators would be willing to place so high an estimate on the percentage of successful cases, of intranasal enlargement of the duct as does Ingals, who, as previously quoted, states that 95 per cent of all frontal sinuses may be safely and successfully entered and drained by means of intranasal instrumentation.

It should be stated, however, in justice to the method in Ingals, that his latest instruments and plan of procedure for entering the frontal sinus through the nasofrontal duct are undoubtedly less dangerous than formerly (Ingals' paper before the New York Academy of Medicine. Section on Laryngology, Dec., 1907), for this operator now passes a guide through the duct into the sinus, next by means of the x-ray determines that the position of the guide is correct, and then, when certain that all is safe, drives the burr, which cuts only on its anterior face, along the guide until the whole course of the infundibulum is enlarged and the sinus is entered.

Unfortunately the most perfect drainage which it is possible to secure by operative enlargement of the nasofrontal duct, does not always thoroughly drain the entire frontal sinus, and this simple procedure of enlarging the nasofrontal duct will not, therefore, cure every case even though of an uncomplicated, acute variety of sinus suppuration. As evidence of this, several anatomists have especially pointed out the fact that the frontal sinus is often partially, and more rarely almost completely divided by osseous septa in such a way as to preclude the perfect drainage of every portion of the sinus, even though the nasofrontal duct be widely open. Thus, Hoeve, in an examination of 100 heads (*Laryngoscope*, July, 1907), states that frequently an internal set of frontal sinuses was normally present, due to the middle sagittal septa being complete. Hoeve further states that occasionally a superior accessory frontal sinus was present in thick skulls, and that frequently posterior and external accessory sinuses were found during these examinations. The writer has seen one posterior accessory frontal sinus, and two external accessory frontal sinuses in cases of secondary operation which he performed because of failure to cure by intranasal method, and he has frequently found the external accessory sinus present during external operations in primary cases that seemed urgently to demand the surgical interference.

It must be clear to every operator of experience that intranasal methods of operation upon the frontal sinus are in accord with but one of the several essential principles of successful surgery, viz., the provision for better drainage; and that even this principle is often violated because of the frequent pocketing of the sinus as a result of the presence of coronal or sagittal septa within. It would seem, therefore, that it may be justly stated that there are at least three classes of frontal sinus suppuration that can not be cured by intranasal methods. These are: 1. Those in which the inner table of the skull arches above the sinus orifice of the nasofrontal duct so closely that, to enlarge this orifice through the intranasal route by means of cutting instruments, would almost certainly endanger the life of the patient because of injury to the dura mater at this point. 2. Those cases in which more or less complete sagittal or coronal bony septa within the sinus prevent good drainage even though a large nasofrontal opening be provided. 3. The cases in which extensive pathologic changes have taken place in the mucous lining or osseous walls of the sinus.

Having briefly shown the limitations of the intranasal method of operation on the frontal sinus, when viewed from the standpoint of surgical principles involved, let us next estimate the value of the external procedure when subjected to examination by the same standard.

By following the external method only can the surgeon hope in any satisfactory measure to carry out the surgical principles of removing in an absolutely thorough manner all the diseased tissue involved. This is the method of certainty in this respect, and not one of guess work. No thoroughly trained surgeon is today at all satisfied that he has performed commendable work in the removal of diseased structures unless he actually sees, and therefore, knows, that he has in nowise disobeyed this first essential of all good surgery. Concerning the dictum that the most perfect drainage should be provided, it will probably be admitted by most operators that the external method of operating on the frontal sinus is superior in every way, for when either the anterior or inferior wall of the sinus is removed, or when both have been resected, as in the Killian operation, every means of access possible is provided for the removal of all the diseased structures which will subsequently interfere with the outflow of exudates. The external method furnishes, also, conditions which are more favorable than any other to the healing of the cavity by means of healthy granulation tissue, because, as will be subsequently stated, if this method of operation provides the best means of thoroughly eradicating the diseased structures, and establishes a thorough drainage for the operated parts, it necessarily follows that the most ideal conditions are provided for a healthy growth of the necessary granulation tissue.

The application of well established surgical principles to the external or intranasal methods of operation for the cure of frontal sinus empyemas seems, therefore, to offer little ground for good argument in favor of the latter plan of treatment, after the disease has long since passed the acute stage, or after it is known in any case that the given sinus consists of a series of compartments rather than of a single, smooth cavity, all parts of which drain, funnel-like, toward the infundibular orifice.

Which of the several methods of external operation is most ideal when viewed from the standpoint of the application of correct surgical principles? The writer has had most experience with the Killian operation, and with the open method. The procedure devised by Prof. Killian seems more in accord with good surgical principles than any other method in which the external wound is immediately closed, and therefore if a primary union of the external wound is intended it should be the method of choice. This operation is also desirable from the reason that a minimum of scar is produced. Although the Killian operation is ideal in many respects, and especially is this true when the anterior ethmoidal cells are diseased, the writer believes, judging by his own limited experience, that when radical surgery is indicated in the cure of frontal sinus empyema, that the desired end is often most certain of attainment by following the so-called "open method" of treatment—that which has been advocated and extensively practiced by Coakley—for by this method greater opportunity is offered the surgeon to provide and maintain throughout the whole course of treatment the most perfect drainage; to foster by the art acquired by his experience the natural growth of the necessary granulation tissue; and should perverse or freakish behavior of the granulation structures at any time become manifest, at once to correct and to restore the same to a satisfactory and normal state.

The probable deformity resulting from the open method, has, of course, been an insurmountable objection to its employment in the treatment of frontal sinus suppuration, but the writer believes that a careful and prolonged experience in dealing with the healing of osseous wounds such as the accessory sinuses, by means of obliterating them with healthy granulation tissue, will in great measure enable the surgeon to avoid ugly and greatly depressed cicatrices. Perhaps nowhere in surgery does experience count for more in securing esthetic results, than in the case of closure of an osseous wound by the open method. The surgical principle will presently be stated that over-pressure will stunt or kill granulation tissue. Hence if the gauze packing which is commonly used in the treatment of the sinus by the open method is inserted with too much pressure the filling-in process is lengthened to such an extent that the epithelial structures will probably cover the surface of the wound before the granulation tissue has risen to the skin level, in which event a depressed scar is the inevitable result. A similar outcome may likewise occur from an undue energy on the part of the surgeon in a too excessive or a too frequent repression of the granulation tissue by means of cauterization. A study of this class of wounds during the healing process, and especially a study of the habits and behavior of granulation tissue is, therefore, a very necessary part of the surgical technic, and should provide a satisfactory means of avoiding ugly scars, in many instances at least.

In a personal experience of more than 500 mastoid operations, and 11 frontal sinus cases treated by the open method, the entire after-treatment of which the writer carried out in nearly every instance, opportunity was offered for rather conclusive observation concerning the behavior of granulation tissue under almost all conditions that are likely to occur during the healing of open wounds. The observations thus made have been of great service to the writer in securing a more rapid closure of the frontal sinus, and in avoiding to a great extent the resulting depression and ugly scars. The conditions under which healthy osseous granulation best thrives was noted, as well as the states of the wound which were most favorable to overactivity and necrosis of the granulation structures. The growth of this tissue in osseous wounds as thus observed seemed often freakish, and its behavior such that a useful interpretation of the same was frequently impossible. Nevertheless, in the majority of instances its development was most natural and vigorous under the following conditions: (a) When the wound was sterile, or at least in a great measure approached a state of sterility. (b) When the granulations were moistened only with the exudates which were present, but were neither submerged in secretion on the one hand, nor too greatly dessicated on the other. (c) When there was at no time a too pronounced or too prolonged pressure from the gauze or other dressing which had been inserted into the cavity. (d) When the primary operation on the sinus had been thorough in every respect, and the operated osseous wound had been left smooth and free from any pockets capable of retaining subsequent secretion.

With a thoroughly prepared osseous wound as above described, clinical observation teaches that the subsequent behavior of the granulation buds may be entirely controlled at the will of the surgeon, who may by means of gauze packing, or the application of caustics, repress the growth of new tissue, if he so desires, until the whole cavity has been covered with epithelial structures, and the wound has entirely healed, leaving a maximum of depression and scar tissue. On the other hand it is entirely possible, in most instances at least, to secure in a satisfactory way, a type of vigorous granulation structure which will rapidly obliterate the sinus by filling it in to a level with the skin structures, which latter are then, and not till then, allowed to bridge across the wound. Healing of the sinus by the open method of treatment will, therefore, leave a minimum of depression and scar tissue, provided the surgeon will exercise that control over the granulation structures which experience has abundantly shown may be exercised almost at the operator's will. The desired pliability of the new tissue is based almost entirely upon the surgical principles of efficient drainage, absence of undue pressure upon the granulation buds, and a properly prepared wound to begin with.

Clinical observation of the processes of the healing of bone wounds by granulation also teaches that if adequate drainage is provided, and the wound is secured against exposure to outside pathogenic bacteria, a sufficient degree of sterility will usually be secured to encourage rapid and successful filling-in of the cavity. The surgical principles involved in the provision of such ideal drainage for this class of wound will also insure against the accumulation of exudates and the consequent harmful flooding of the new tissue. In sinus surgery, therefore, successful restoration of the cavity, or its complete obliteration, needs scarcely be expected in any case in which the external wound is immediately closed, unless the operator is absolutely convinced that the drain canal he has provided is ample to insure against retention of fluids and the inevitable infection of the same, should they be permitted to stagnate. Even though such an ideal drainage is established at the time of the operation, if immediate closure of the wound is practiced two things may speedily happen to cause failure to cure. Those are, 1st, the too rapid closure of the drain canal by granulation tissue, and, 2nd, the formation of pockets through faulty behavior on the part of the granulation and the consequent retention of pus in the sinus, even though an adequate drainage canal remains intact. This latter condition is one which, in the writer's opinion, is responsible for the failure in many cases which have not improved, and in which secondary operations have, therefore, been necessary. It has already been stated that the behavior of granulation tissue, even when all conditions are apparently normal, is sometimes freakish and unaccountable. For example, the writer has often observed during the post-operative treatment of open wounds in the several head sinuses, that occasionally when all had been progressing perfectly well, if the patient be not seen for 36 or 48 hours, granulations of unaccountable size would spring up in some part of the wound and grow toward similar granulations in another part with such amazing rapidity that adhesion between the opposing buds took place, a wall was constructed across the healing cavity, and, before the surgeon was scarcely aware of what had occurred, some portion of the wound was already badly drained, the granulations rank and perhaps necrotic, and unless the faulty conditions thus established were promptly corrected, failure to heal the wound was inevitable. Of course, should such an occurrence take place in an open cavity, every part of which can be inspected at any time, failure should not result, because the error is easily detected and corrected; but when the external wound has been immediately closed at the time of the operation, or when intranasal drainage only has been practiced, the surgeon can only surmise what is going wrong, and is, therefore, helpless to correct any freaks of healing by granulation, except by means involving a secondary operation.

XXX.

A CASE OF ACUTE SUPPURATION OF THE LABYRINTH FOLLOWING ACUTE OTITIS MEDIA—OPERATION; RECOVERY.*

By Edward Bradford Dench, M. D.,
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While cases of acute suppurative inflammation of the labyrinth are not so very uncommon, the symptomatology of the affection is far from clearly defined. This especially is true of those cases which follow an acute inflammation of the middle ear.

I take the liberty, therefore, of reporting somewhat in detail the following case, on account of the rather unusual manner in which the symptoms presented themselves.

The patient was a man sixty-five years of age who, about two weeks before I saw him, had an acute attack of grippe, with an acute suppuration of the right ear. Spontaneous perforation occurred early, followed by profuse discharge. The drainage from the ear was insufficient, however, and eleven days before I saw him, the right drum membrane was incised by a competent otologist. Free drainage was established, but the patient had suffered ever since the beginning of the attack, with severe pain in the head, together with pain in the right mastoid region. The patient also complained of inability to sleep. This sleeplessness, however, could not be attributed to the acute inflammation in the right ear, as he had been suffering from insomnia for some time prior to the acute otitis.

When I first saw the patient, I found him rather excitable, complaining of severe pain in the head, particularly upon the right side. There was some tenderness over the mastoid antrum, over the tip of the mastoid, and over the mastoid emissary vein. This tenderness was elicited only on firm pressure, and was not very severe. There was a profuse, purulent discharge from the ear. Examination revealed a perforation in the antero-superior quadrant of the drum membrane, just beneath the anterior fold. A probe introduced through this perforation detected roughened bone. The aural discharge was not examined at this time, but it had been examined by the attending physician, who reported that the smears showed a diplococcus, and that no streptococci were present. A blood count had been made, showing a normal leucocytosis. No differential count had been made.

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* Read before the Annual Meeting of the American Otological Society, Atlantic City, June 24, 1908.

When I first examined this case, I was inclined to believe that operative interference would be necessary. The temperature of the patient was practically normal at this time, and it had been so for several days prior to my first visit. As I have said, when I first saw the patient, I believed that immediate operation would be necessary. I saw the case frequently, however, and the mastoid tenderness disappeared. The discharge from the meatus diminished somewhat in quantity, but still retained its thick, purulent character. The patient was observed, at intervals, for one month. During this time the mastoid tenderness entirely disappeared, as did also the headache and sleeplessness. The patient was able to be about and take regular out-of-door exercise. When, however, he attempted any mental exertion he became very nervous and easily fatigued. Owing to the fact that the purulent discharge from the ear still persisted, in spite of the fact that all mastoid tenderness had disappeared, I determined to open the mastoid. This operation was performed one month after I first saw the patient. At the time of operation the cortex was found to be perfectly normal, and fully an eighth of an inch in thickness. On opening the cortex the entire mastoid process was found to be broken down and filled with granulation tissue. The internal table was entirely destroyed, and the lateral sinus ran through a mass of granulation tissue. The wall of the sinus had become softened, and the sinus was opened through the gentle removal of these granulations by means of the curette. Hemorrhage was easily controlled by means of gauze packing. The complete mastoid operation was done. A small epidural abcess was found just at the knee of the sinus, containing about half a drachm of thick yellow pus. After the mastoid operation, the patient complained of severe pain in the head, which prevented him from sleeping, and required moderate doses of sedatives. I attributed this pain to the blocking off of the lateral sinus, but owing to the extensive exposure of dura, and the fact that the case had progressed about six weeks prior to the operation, I also feared the possibility of a collection of pus within the cerebellum. All unfavorable symptoms, however, gradually disappeared, and the patient was able to leave the hospital and return home. The wound did well from the first, and in about five weeks after the operation, had contracted down to a comparatively small sinus, through which a probe could be passed into the middle ear. The discharge from the ear ceased almost immediately after the operation, and the ear remained dry for about two weeks. At this time, the patient contracted a slight cold, when the discharge from this external auditory canal reappeared and persisted. There was free through-and-through drainage from the posterior wound to the canal, but, in spite of this, the discharge from the canal never completely disappeared. About five weeks after the mastoid operation the patient was suddenly seized with a severe attack of vertigo. In walking there was a tendency to fall toward the unaffected side. This vertigo was attended with nausea and vomiting. When the vertiginous symptoms first appeared, there was no nystagmus, but twelve hours later there was pronounced nystagmus, this being more marked when the patient looked toward the healthy side, although there was slight nystagmus on looking toward the affected side. This latter symptom, however, disappeared quite rapidly, and nystagmus could then only be elicited by looking toward the healthy side. The temperature was normal on the night of the first appearance of the vertiginous symptoms, and the next day it reached 101°. The patient complained of considerable headache.

It should be stated that immediately after the mastoid operation several blood counts were made, and at no time did the polymorphonuclear count rise above 68%, while on most occasions it was below 60%. Immediately following the acute vertiginous symptoms, the polymorphonuclear count rose to 74.5%, with a leucocytosis of 13,400. A smear from the wound showed a few bacilli, a few diplococci without capsules, and some large and small cocci. The discharge did not seem to exhibit any particular virulence. The patient was seen in consultation by a prominent neurologist, who was at first inclined to believe that there was an abcess in the cerebellum. The reflexes at this time were somewhat exaggerated, and there were decided symptoms of meningeal irritation.

Knowing that all these symptoms could have been occasioned by an acute invasion of the labyrinth, I refrained from any operative interference. Forty-eight hours after the appearance of the labyrinthine symptoms, the temperature became normal. The polymorphonuclear percentage dropped to 56.4% and the leucocytosis fell to 9,400. An examination of the fundus oculi simply showed some slight tortuosity of the veins. I might say that this same condition of the ocular fundus existed, a few weeks after the simple mastoid operation had been performed. As the examination was in each instance, made by the same man, he reported that there had been practically no change in the fundus oculi. Owing to the fact that the temperature had fallen, and that the blood count showed no indication of absorption of pus, and also, as the patient's symptoms were gradually disappearing, I decided to defer operation.

The patient gradually, steadily improved for the next six weeks. At the end of this time the mastoid wound had entirely closed, with the exception of a small sinus at the upper angle of the wound which led directly into the middle ear. The discharge from the ear, however, continued, and the probe introduced through the perforation in the drum membrane, still encountered roughened bone. An examination of the discharge from the ear showed streptococcus capsulatus.

During the six weeks following the vertiginous attack, the symptoms of disturbed equilibrium steadily improved. The vertigo now was so slight as to cause the patient but little annoyance. The nystagmus entirely disappeared, as well as the pain in the head, and the patient was able to sleep well at night. On account of the persistence of the discharge, however, I determined about seven weeks after the attack of vertigo, to perform the radical operation. At the time of the operation, I found a small eroded area in the horizontal semicircular canal. The lumen of the canal was opened, and a small amount of turbid fluid escaped. The oval window was then examined and, in order to establish free drainage of the labyrinth, was enlarged. In enlarging the oval window, a scale of necrotic bone was removed from the promontory, uncovering the lower and a portion of the second turn of the cochlea. The interior of the cochlea presented a reddish appearance, as though some granulation tissue was present there, although no pus could be detected. Thorough drainage was established between the opening in the horizontal semicircular canal and the opening in the vestibule below the aquaeductus Fallopii. The posterior wound was completely closed, with the exception of a small opening at the lower angle, where a gauze drain was inserted. The external auditory meatus was enlarged by cutting a tongue-shaped flap from the concha, thus securing complete drainage of the entire cavity through the meatus. A meatal skin graft was applied.

Since the radical operation the patient has progressed favorably. The dizziness has entirely disappeared, there has been no nystagmus, and at the present time the radical cavity has completely dermatized.

The examination of the ear after the appearance of the vertiginous symptoms was interesting. A moderate whisper was heard, two weeks after the vertiginous attack, at 7 feet from the ear. The tuning-fork placed on the forehead, was heard better on the affected side, but the absolute bone conduction was diminished on both sides. The upper-tone limit was lowered to 4 Galton on the affected side. One month after the attack, and before the radical operation, a moderate whisper was heard at 5½ feet in the affected ear. Bone conduction was about equal on both sides, but the tuning-fork placed upon the forehead, was referred to the affected side. The lower-tone limit in the affected ear was elevated to 512 double vibrations per second. The upper-tone limit was 4 Galton. The tone limits on the unaffected side were normal.

After the operation, moderate whisper was heard at a distance of 6½ feet, and the vibrating tuning-fork placed upon the forehead was referred to the affected ear, although absolute bone conduction on the affected side was slightly diminished. The lower-tone limit on the affected side, was 512 double vibrations and the upper-tone limit was 4.5 Galton.

This case to me was interesting from several points of view: In the first place, the appearance of labyrinthine symptome, in spite of free drainage of the middle ear through a posterior opening. We usually expect to find labyrinthine symptoms developed only in cases where there is some obstruction to drainage. In this case, the middle ear was draining freely, not only through the mastoid opening, but also through the external auditory meatus, but in spite of this, there was an invasion of the labyrinth. Whether this invasion took place through the oval window, and the carious area in the horizontal semicircular canal occurred secondarily, or whether invasion took place through the horizontal semicircular canal, can only be a matter of conjecture.

A second point of interest was the relatively short duration of the acute labyrinthine symptoms. At the end of forty-eight hours from the inception of these symptoms, the temperature was normal, and remained so in spite of the fact that the labyrinth was the seat of suppuration. The gradual disappearance of the labyrinthine symptoms, such as vertigo and nystagmus, is also a matter of interest.

When we come to examine the tuning-fork tests, the fact that the tuning-fork held upon the forehead was always referred to the affected side, is a phenomenon rather difficult of explanation. With destruction of the labyrinth, we would naturally expect that the tuning-fork would be referred to the opposite side, and that bone conduction on the affected side would be greatly diminished. My case shows, however, that five or six weeks after the labyrinthine symptoms first appeared, the tuning-fork placed upon the forehead, was still referred to the diseased ear, and that the upper-tone limit was not greatly lowered. Bone conduction, however, on the affected side was slightly diminished. Even after the radical operation, we still find the tuning-fork reaction somewhat confusing. The vibrating-fork placed upon the forehead, in the median line, was still referred to the affected ear. Bone conduction on the affected side was slightly diminished,—not absolutely lost, while the upper-tone limit was only reduced to 4.5 Galton. The elevation of the lower tone-limit would simply be indicative of the middle ear lesion and would have no bearing upon the labyrinthine condition.

It is a question in this case, whether it would have been wiser to have operated when the labyrinthine symptoms first appeared. From the satisfactory outcome of the case, I am inclined to think that I pursued the preferable course. Had I operated at the time of the acute symptoms, I rather think that I might have opened up a fresh avenue of infection in the labyrinth. Had the fever persisted, I should naturally have exposed the horizontal semicircular canal and the inner wall of the middle ear at once, by the radical operation; but in the absence of febrile symptoms, I felt that it was better to wait until the diagnosis of labyrinthine involvement could be definitely established. Had I operated within the first twenty-four hours after the appearance of the symptoms, I should probably have explored the cerebellum, as the symptoms pointed very strongly to involvement of the cerebellum. Such an exploratory operation would, of course, have been unnecessary, and might have been followed by serious results.

While it is impossible to lay down any definite rule in regard to these cases, I believe that if the temperature does not rise above 101° or 102°, it is well for us to wait for a certain interval, and see if the labyrinthine lesion will not be self-limited. If, however, the temperature continues to rise, it is necessary to operate immediately, as then, the only hope of preventing an extension of the inflammation to the cranial cavity, is by prompt operation. This has been demonstrated by Jansen and others.

I think the rule should be, therefore, that, with acute labyrinthine symptoms and pronounced elevation of temperature, operative interference should be at once instituted. With acute labyrinthine symptoms, however, with little or no febrile movement, it is wiser to wait for a few days at least, before opening the labyrinth.

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XXXI.

BLACK HAIRY TONGUE (LINGUA VILLOSA NIGRA).

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The disease, for which some German authors use the characteristic name "Schwarze Haarzunge" (Black Hairy Tongue), and which consists in the growth of long, dark, hair-like structures on the surface of the tongue, is often thought of only as a curiosity. However, the affection is of some interest, since it sometimes happens that the patient believes the disease is of a cancerous nature, and because it very often puzzles the physician on account of its resistance to treatment. Moreover, the condition certainly is not as rare as is generally considered. The etiology and the pathogenesis of the disease are very obscure. In consequence of this, the disease has been given many different names. Some of these are merely the expressions of the authors regarding the nature of the disease.

In the literature I have found the following names:

The older ones—Lingua nigra or Nigrities linguae; English—Black Tongue or Black Hairy Tongue; German—Schwarze Zunge, or Schwarze Haarzunge; French—Langue noire pileuse or villeuse.

Later ones—Hypertrophie epithéliale piliforme (Féreo[29]), Melanotridria linguae (Augier,[4] Maraval,[53] Mracek,[53]); Pseudomelanosis linguae (Birch-Hirschfeld[8]); Lichenoid (Butlin[17]) and Hyperkeratosis (Brosin[14] and others).

The investigators who have considered the disease of parasitic origin have given it the following names:

Glossitis parasitica (Hofheimer[35]); Keratomycosis (Roth[79]); Glossophytie (Pasquier[68]); Mycosis linguae mucorina nigras, Nigrities mucorina linguae (Ciaglinski and Hewelke[20]) and Mycosis mucorina (Sendziak[89]).

It is easily understood how this redundant nomenclature has caused some embarassment. For instance it is seen that Goodale[32] distinguishes the cases of black tongue which he describes under the head of "Hyperkeratosis lingualis" from those cases in which moulds are found, to which he gives the name "Lingua nigra."

The best designation, in my opinion, is the name that corresponds to the German "Schwarze Haarzunge" and the English "black hairy tongue," viz., "Lingua villosa nigra." This name has the advantage of including the two most characteristic features of the disease: viz., the dark color and the hairy appearance of the tongue.

The first to observe the condition was (according to Arnold) Amatus Lusitanus[51] who, in 1557, saw on the tongue of a man "hairs which came again after they had been pulled out." Later Rayer,[71] in his "Traite des maladies de la peau," 2nd edition, 1835, writes that sometimes there is seen a black color on the back of the tongue, and he names the phenomenon "Negrities." In the same year an anonymous correspondent in the "*Boston Medical and Surgical Journal*"[10] states that his own tongue is black. In 1852 the disease is mentioned by Hyde Salter[92] in Todd's "Cyclopedia of Anatomy and Physiology" and in 1853 Eulenberg[27] wrote a paper entitled "Ein schwarzen Zungenbelag" in which he speaks of the case of a child whose tongue was black far down into the throat. It is doubtful if this was a typical lingua villosa nigra. The same may be said of four cases which Bertrand de St. Germain[91] describes in the year 1855. This author is of the opinion that the black color is due to a pigmentation of the same kind as is found in the skin of the negro. He does not mention the hairy growth which is characteristic of the disease. In 1869 Raynaud[73] published his classical paper "Une nouvelle affection parasitaire de la langue." In this we find for the first time a description of the microscopic appearance of the disease. In the same year, Gubler[33] gave a description of the disease in the "Dictionnaire encyclopédique des sciences médicales." Since that time, the publications have become quite numerous. Several theses concerning this subject have appeared, among which are those of Laveau[46] (1875), Dessois[25] (1878), Ch. Rayer[72] (1883), and Boucher[11] (1903).

I have found in literature 88 different authors (their names may be found in the bibliography) who have written papers on black tongue, and there are probably many more who have escaped my notice. In all, I have found published 128 cases of black tongue, to which I can add ten cases that I have treated in the Dispensary for Ear and Throat Diseases in the Municipal Hospital of Copenhagen.

The following is the result partly of the investigations of the ten cases examined by me, and partly of the cases in literature.

THE APPEARANCE OF BLACK TONGUE.

The typical black tongue presents an appearance as follows: On the back of the tongue of an otherwise healthy individual is found a brownish-black spot. The spot is located just in front of the circumvallate papillae and extends forward to about one inch from the tip of the tongue. The spot is symmetrically arranged on each side of the median line and the borders and tip of the tongue are covered with a whitish fur. The spot owes its appearance to long hairs, that is, the prolonged papillae filiformes. These have a length of ¼ to ½ inch and are longest in the posterior part and on both sides of the sulcus medianus linguae. In this way the middle part of the tongue may appear very deep, as though there was a defect there. The surface of the tongue can well be compared to the appearance of a wet, long, black-haired dog, or to a wet silk hat. The hairs are directed from behind forward and if the tongue is rubbed in the opposite direction the appearance just mentioned is noticed. The surface of the tongue is somewhat slimy and on the base and in some places between the papillae vallatae, there is a whitish fur that resembles thrush. When the black spot is scraped with a curette the prolonged papillae are very easily cut off and do not leave a bleeding surface. These papillae are conical in shape and are black only at the apex of the cone. At the base they are thick and gray. They float in an opaque, mucous fluid.

In black tongue, there are these two characteristics to be found, viz., (I) more or less hypertrophy of the papillae filiformes, and (II) the dark color of the papillae. Most of the cases in literature show these two peculiarities and can be grouped under the head of lingua villosa nigra. All cases which do not show these marks should not be given this name. Consequently, the cases described by Curtis,[23] Krimer,[40] Eulenberg,[27] and Bertrand de St. Germain[9] cannot be regarded as genuine linguae villosae nigrae. Naturally there may be some difference in the appearance of the cases within the said limits.

We may at times find some difference in the nature of the color. Although the color commonly is black or brownish-black, a yellowish-brown color has been found. In one case, the tongue had a dark greenish-gray appearance, while in another the spot was black in the center and green on the borders. In still another there was a white center with a dark circumference. Sometimes, it has been noticed that the color changes. In one case of Rydygier's[81] the tongue was at first black, but later in the disease turned blue. In the case described by Mickulicz and Michelson[59] the tongue was at first blue but later when the stain had disappeared it presented a whitish, furred appearance due to the long papillae. Yet, in all these cases the results of the microscopic examinations were similar. It is therefore probable that there has existed an analogous condition in all of these cases.

The form, position and size of the colored spot may vary. As a rule the spot is symmetrically arranged along the median line of the tongue just in front of the papillae vallatae, but it may be found on one side or at the tip of the tongue. It may be as small as a one-cent piece or it may extend over the entire surface of the tongue from the tip to the circumvallate papillae and to the borders on either side.

SYMPTOMS OF BLACK TONGUE.

In many cases, the disease gives no symptoms and is discovered accidently by the patient or the physician. In other cases, many disagreeable and severe symptoms are present. In my opinion, there are many cases in which the black tongue is not responsible for these symptoms, namely, those cases in which the patient besides having this disease has suffered from a more serious trouble which latter may be the cause of the symptoms. Only in those cases of black tongue affecting otherwise healthy individuals and where the symptoms appear and disappear with the disease, can one safely say that these symptoms are due to the disease. Considering these facts, it cannot be said that one severe symptom is actually due to the black tongue. The symptoms following this disease are all local: dys- and paresthesia of the tongue; sensation of dryness, tickling, etc., in the mouth and throat; sometimes pain in the tongue or the gums; in some cases the sense of taste is weakened, while in others it is increased to such an extent that some foods produced the sensation of pain. In some patients the disease produces fetor ex ore.

COURSE OF BLACK TONGUE.

The disease usually develops in the following manner: A white spot first appears. This spot is due to the prolonged, hypertrophied papillae filiformes. It subsequently is stained a grayish-brown and later becomes black or brownish-black. This sometimes takes place in a very short time, sometimes over night (Schmiegelow,[86] Ciaglinski and Hewelke[20]). In one of my cases, the development of the black color covered a period of about four days. In some cases the course of the disease is very short, the color disappearing in from two to seven days. In others, however, the course is more chronic and may extend over a period of several months or years. In one case reported by Sell[87] the duration of the disease was fifteen years. Often, perhaps in all chronic cases, the disease is intermittent, appearing and disappearing at intervals. Out of 48 cases reported in the literature in which the duration is mentioned, 31 lasted one year or more, while the remaining 17 lasted from two days to three months. The most interesting fact in regard to the course of the disease is the relation that exists between the black tongue and the "principal" disease of the patient. The acute cases of black tongue are found on patients suffering from some acute affection (acute gastro-enteritis, pleuritis, etc.), while the chronic cases commonly occur in patients who have some chronic disease (tabes, chronic constipation, etc.). Black tongue is only a symptom that in certain conditions accompanies different acute and chronic diseases.

PATHOLOGIC ANATOMY OF BLACK TONGUE.

Former investigators simply curetted off the papillae and, after putting them in potash, glycerine or acetic acid, and treating with methylene-blue or Bismark-brown, have examined them microscopically. I have examined specimens from five patients, treated by me, in the following manner: The papillae were curetted off from the tongue, hardened in alcohol, imbedded in paraffin, cut on the microtome and examined both unstained and after staining by the Van Giesen method. The advantage of this method of staining lies in the fact that the keratosed cells are stained a reddish-yellow and the epithelial cells a bluish color. I have also used the Ziem-Nielsen tubercle stain and have been able in this way to bring out the horn cells. By this latter method, the keratosed cells are stained red while the ordinary epithelial cells are deprived of their color by the sulphuric acid. These, however, can be stained later by using methylene-blue. To show the keratohyalin and eleidin granules. I used the v. Ebbinghaus method and the haematein coloring (Unna). In staining the microbes, I used partly the Gram and partly the Weigert method. In all the examined cases the microscopic appearance was about the same. The following is a report of the microscopic examination of papillae from the tongue of a man 70 years of age, who was suffering from progressive bulbar paralysis and an intermittent black tongue: The papillae filiformes are very long (some of them three-fourths of an inch), the most of them being very thin and hair-shaped, while the others are thicker and conical. The epithelial cells on the surface of the papillae are arranged like tiles on a roof. They are hair-like and are loosened at their proximal ends. On this account, they resemble an inverted ear of corn. Commonly, two papillae are attached to the same base, and are connected by hair-like keratosed epithelial cells. The center of the papilla is made up of polygonal epithelial cells, which are stained bluish by the Van Giesen method. The cells have a well-stained nucleus. The surface and tip of the papilla is made up of a closely striped layer of cells which stain a reddish-brown with the Van Giesen method. These are pointed, spindle-shaped keratosed cells which have more or less degenerated nuclei. It is the most superficial cells of this layer which, being loosened at their proximal ends, and projecting out from the papilla, give it its corn-like appearance. Keratohyalin and eleiden granules are not to be found. In the unstained specimens it can be seen that the dark color of the papilla is due to the epithelial cells which have a diffuse brownish appearance. This color is darkest at the tip. Between the papillae can be seen numerous free epithelial cells, some of which are keratosed and have a faintly stained nucleus and reddish-yellow protoplasm (Van Giesen) while others have a pink protoplasm and a distinct nucleus.

On the surface of the papillae, numerous microbes can be found, most of which are round or slightly pear-shaped clear bodies with a high refractive power. They are all about the size of the nuclei of the epithelial cells, and are arranged in irregular layers both on the surface of the papillae, where they are attached to the barb-like cells, and at the center of the papillae between the epithelial cells. They are readily stained by the Weigert and the Van Giesen methods and are probably spores of mould. Many slightly curved bacilli, some diplococci and some coccobacilli are found, but no leptothrix.

As was said, the papillae in all cases showed similar microscopic appearances. The variations mostly were in the length and thickness of the papillae and the kind and number of the microbes. The large, round, clear microorganisms mentioned above were found in only one case. Bacteria, varying in numbers, were always found. The number depends in a great part on the surface of the papillae. Where there are many, long, barb-like epithelial cells on the surface, many microbes are to be found. Where the epithelial cells lie close together, few microbes are found. The papillae in all cases were connected at their bases. It is therefore not true, what some authors (Mourek,[62] Brosin[14]) maintain, that in black tongue the papillae are always bound together in thick conical bundles.

In all the cases which I treated, I tried to cultivate mould from the tongue. The papillae were scraped off with a sterilized curette, and with a sterile glass tube I rubbed some on a mixture of wheat-bread with 10 per cent gelatine and on malt agar. Some of these were put in an incubator at 24° C. and the others at 37° C. In all cases, colonies of white and gray microorganisms developed but no black or brown moulds.

ETIOLOGY OF BLACK TONGUE.

1. Sex. In the 128 cases of black tongue reported in the literature and the 10 of my own, making 138 in all, 12 were in children below 15 years of age, 56 were in men and 24 were in women. The reports of the other 46 patients were not at my disposal. From this it is seen that the disease has occurred in twice as many men as women. This is no doubt due to the etiologic importance of tobacco (see below).

2. Age. The ages of only 79 patients were at my disposal. These I give in the table below:

Age	Men	Women	Sex Unknown	Total
10 to 14 years of age..	4	3	2	9
15 to 19 years of age..	1	1	..	2
20 to 29 years of age..	13	4	..	17
30 to 39 years of age..	7	2	1	10
40 to 49 years of age..	9	2	..	11
50 to 59 years of age..	6	1	..	7
60 to 69 years of age..	5	3	..	8
Over 70 years of age..	7	8	..	15
Total	52	24	3	79

(Among those numbered under "over 70 years of age" are the ones mentioned in literature as "old.")

From the above table it is clear that the assertion of most authors that black tongue is found almost exclusively in old individuals does not hold good. While there are 30 patients of more than 50 years of age, there are nine cases in children and 40 in persons between 15 and 50 years of age, and among these last there are 17 whose ages range from 20 to 30 years.

3. Different local and general diseases as etiologic factors: In 13 of the reported cases the patient was otherwise perfectly healthy. Two of these were children. In all the other cases, the patient was suffering from some other severe disease. Twelve of the patients had syphilis and of these 2 were suffering from stomatitis, 3 had papules in the mouth and 2 were excessive tobacco smokers. Seventeen patients had different affections of the mouth, pharynx or larynx (of these only 1 was more than 70 years of age, the others were less than 50). Nineteen patients had various affections of the stomach or intestines. Twenty patients were in a state of great weakness either on account of some severe acute trouble (scarlatina, typhoid fever, influenza, etc.), or of some grave chronic disease (various nervous diseases, tuberculosis, etc.), or of old

age. Other intercurrent diseases reported were: Nervousness, 3 cases; asthma, 1 case; pleuritis, 1 case, emphysema pulmonum, 1 case; epilepsy, 1 case; stricture urethrae, 1 case, and diabetes. Of these 8 patients, 5 were more than 60 years of age.

4. The more intimate etiologic factors and the pathogenesis: During the past years many theories have arisen as to the origin of black tongue. Hutchinson[37] maintains that all cases are due to an illusion or to an accidental coloring of the tongue. The other authors may be divided into two groups, in regard to their opinions concerning the etiology of the disease. One group claims that the disease is due to microorganisms while the other maintains that these do not play a part in the development of the affection. There are also differences of opinion among those of the first group regarding the pathogenesis of the disease. (a) Some say that black tongue develops from a black mould which grows on the surface of the tongue. (b) Others have the opinion that microorganisms first produce an hypertrophy of the papillae and then produce the black color. (c) A third group believes that microorganisms produce an hypertrophy of the papillae but do not produce the stain. (d) The last group maintain that the black color is due to the microbes but that the hypertrophy is not.

(a) Ciaglinski and Hewelke[20] were the first who were of the opinion that black tongue was caused by the growth on the surface of the tongue of black moulds and, later on, Schmiegelow[86] and Sendziak[89] were of the same opinion. In the year 1893, the two first named investigators succeeded in cultivating a black mould from the surface of a black tongue which had developed acutely in a patient suffering with influenza. The mould grew at 15° to 25° C. on potatoes, bread, and particularly well on wheat bread with 5/10 per cent gelatine. At 37° C., it could not be cultivated. The growth appeared as an umbrella-shaped columella on which developed spores, which were first white and later became black. Ciaglinski and Hewelke called the mould Mucor niger. The next year (1894) Sendziak[89] cultivated a similar fungus on wheat bread gelatine; it was taken from a black tongue of the usual typical appearance. However, the growth differed from that described by Ciaglinski and Hewelke in two respects: (a) it did not grow on potatoes, and (b) it had an oval, not an umbrella-shaped, columella. From another case, one of brown tongue, Sendziak[89] cultivated a similar mould, whose spores were brown or yellow. Later (1905) Sendziak[89] states that Paltauf in Vienna found in a patient suffering with black tongue and peritonsillar abscess (also, icterus, enteritis, and fever) Mucor corymbifer both on the tongue and in the pus from the abscess.

Schmiegelow[86] has cultivated moulds from two cases of black tongue. The first case was a chronic black spot on the tongue of a man 42 years old. The microscopic appearance was of the usual nature, hypertrophied papillae filiformes, etc. He found, however, in this case two different types of mould: (a) a chocolate-brown hyphomyceta resembling Oospora ruberrima sacc. and (b) a dark mould, Trichosporium chartaceum (Pers.) sacc. which he (Schmiegelow) regards as pathogenic. The second case was an acute black tongue which developed over night in a lady 70 years of age who was suffering with stomatitis and acute gastro-enteritis. The black spot was situated on the front part of the tongue and had the usual microscopic appearance. Cultures on wheat-bread-gelatine after a week produced colonies of a brown color made up of the mould, Hormodendron cladosporides. Schmiegelow thus concludes that black tongue is produced by the growth of a black mould on the surface of the tongue and he maintains that the reason that other investigators have not been able to find this mould is that they have not used the proper cultivating medium, viz., wheat-bread-gelatine.

All other investigators who believe in the parasitic theory recognize that black tongue depends upon (1) an hypertrophy of the papillae filiformes and (2) upon the black stain of these. Some of these are of the opinion that both the hypertrophy and the black color are produced by parasites. Among these is Maurice.

Raynaud,[78] (1869) who was the first to find microorganisms in black tongue and who was the first to give a careful description of the microscopic pathology of the affection, found (in two out of four cases) on the surface of the papillae, large quantities of round or oval bodies with high refracting power varying from 3/5 micra in size. Though he did not find any mycelia, he considers the bodies to be spores similar to trichophyton, and holds them responsible for the affection.

In 1878 Dessois[25] in his thesis describes similar bodies. He had examined three cases of black tongue. In the first case he found small bodies of the same size as those Raynaud described but they were arranged in straight lines vertical to the surface and not in irregular heaps as were Raynaud's spores. In the second case he found in some places small heaps of spores which were somewhat larger than those in the first case. In the third case he found spores in isolated groups on the hairs and having an average size between those of the first and second case. In spite of the different microscopic appearances and although autoinoculation gave a negative result, Dessois believed that he had found the specific microbe of black tongue and called it glossophyton. He believed that the microbes located on the base of the papillae, by their irritation stimulated the growth of the papillae and that later they produced the stain.

Schech,[83] in his text-book of 1885, joins those believing in the parasitic theory, based on the publication of Dessois. Two years later, however, he had the opportunity to examine a case of black tongue but did not find any spores and consequently abandoned the parasitic theory, which he opposed in the next edition of his text-book[84] in 1896.

Lannois[45] (1888) was of almost the same opinion as Dessois. In two cases of black tongue he did not find any microbes but in a third case he found round or oval colorless refracting bodies which looked like spores and which did not stain with the analine dyes. Attempts to cultivate them were unsuccessful. Lannois maintains that a local disturbance of nutrition prepares the ground after which the bacteria become the decisive cause of the development of the hypertrophy and the coloring of the papillae.

(c) Some authors maintain that the hypertrophy but not the coloring is produced by parasites. This theory was brought forth in 1887 by Roth.[79] He describes two patients whose tongues were covered with hypertrophied papillae filiformes. In the first case the color was white while in the second it was black, the tips of the papillae being stained (by tobacco). The author did not succeed in making a pure culture of the microbes found, among which were sarcina, oidium albicans and leptothrix. Roth believes that the hairy structures are produced by the usual microorganisms of the mouth and names the condition keratomycosis.

David[24] assumes that the cause of the hypertrophy is the irritation of the bacteria which are located at the base of the papillae and that later they grow and cover the latter as a cloak. Rostowzew[73] is of this opinion also and on cultivation found a cladothrix and a sort of yeast. Inoculation of man and animals was unsuccessful.

(d) There are still some who believe that the color but not the hypertrophy is produced by parasites. Most of these have not examined cases personally but rely on the investigations of others.

Cornil and Ranvier[22] are of the opinion that the black color is produced by myriads of black micrococci. These they believe to be the same as are usually found in the mouth, differing only in color. These are arranged in zoogloea as numerous small round immobile bodies and are intensely stained with the analine dyes.

Butlin[18] attributes the hypertrophy to the fact that the friction of tongue is less in front of the papillae vallatae and he believes the color to be due to microorganisms.

It seems as if Vignal had the same opinion. He relates that Malassez has cultivated the same spores as Dessois[25] found, and that he had further found similar spores in 18 cases of "white tongue." Dessois assumes that the black and the white tongue are produced by two varieties of the same microbe, a white and a black one. These microbes which Dessois named glossophyton are in Vignal's opinion identical with the alphacoccus described by him.

Finally there are several authors who believe the affection to be of parasitic origin but do not mention anything particularly about the pathogenesis. Hofheimer[35] names the disease glossitis parasitica. Papon[66] suggests that the cause of the reappearance of the disease is the resistance of the parasites. Gaston and Nicolau[31] believe that black tongue is produced by a variety of leptothrix and Boecher[16] also is inclined to consider the parasite as being a leptothrix in the early stage of development. Pasquin[68] found the spores described by Dessois. Leviseur[49] is of the opinion that the dark color of the papillae is produced by (1) drying, (2) chemical influence of food, (3) certain bacteria which act as chemical agents, and (4) certain drugs. He found sulphur but no iron in the black stain and considers it identical with the color in the keratosed cells (Unna[110]).

(3) Those who do not believe in the theory that microbes are etiologic factors in black tongue have many different opin-

ions concerning the etiology and pathogenesis of the disease.

Armaingaud[2] believes the cause is a disturbance of the vasomotor nerves as is seen in the chromidrosis of the skin.

Solis Cohen,[21] Chevalier,[19] Mourek[62] and Masoin[54] maintain that black tongue is due to trophic disturbance, and the last named compares the affection to papilloma.

Hyde Salter[82] enunciated the theory that black tongue is due to pigment granules which are identical to those found in the scrotum at the age of puberty, when the skin is filled with blood. Matthieu[56] assumes that "des granulations graisseuses dans les gaines épithéliales" contribute to the dark color and finally Goodale[32] found on the surface of the papillae refractant granules of a yellowish-brown color together with characteristic bacterium (probably large cocci or spores). It is interesting from an historical standpoint to note that Rayer[71] (1835) mentions in one form of black tongue a "coloration pigmentaire" by which he distinguishes it from the artificial form due to food and drugs.

It is only by a few, and especialy the older authors, that pigment granules are reported to have been found in cases of black tongue, so that we can discard the theory which claims all cases of the disease to be due to pigment granulae. Wallerand[96] claims that these observations were wrong.

Most of the investigators during the last few years are of the opinion that black tongue is due to a hyperkeratosis of the epithelium of the papillae filiformes. This theory owes its origin to Schech[85] (1887). He found that the papillae cut from the tongue were completely keratosed and he maintains that the affection is due to an hypertrophy of the papillae filiformes with keratinization and pigmentation. Brosin[14] is also of this opinion. He declares that the affection is due to a genuine hyperkeratosis connected with a consecutive degeneration atrophy (brown coloring) of the cells. Others that join in this theory are Dinkler,[28] who compares the formation of the pigment in the horn-cells of the papillae with the pigmentation of the horn-pearls in epithelioma; Wallerand[96] who compares black tongue with psoriasis linguae, and Mracek,[63] who considers the affection to have the same pathogenesis as pachydemia laryngis. Vollmer,[103] Maraval,[53] Johnston,[38] and Angier[4] also consider black tongue a hyperkeratosis. Only Maraval of all the investigators found fine granules in the epithelial cells. These granules were stained pink with hematoxylin and were also stained by the Gram's method. He considered them to be eleidin.

We must base our opinion as to the true pathogenesis of black tongue on a consideration of all the reported cases and on the different theories as advanced by the different investigators.

First let us consider the parasitic theory: This theory may be supported by different means—(1) microscopic examination and cultivation of the microbes; (2) inoculation; (3) cases of infection.

By microscopic examination and cultures, sometimes numerous bacteria are found. These are for the most part the same as are found in healthy mouths (David[24] found in normal mouths 19 different kinds of bacteria and Mills[108] found 25). Among others the following microorganisms have been found in cases of black tongue: leptothrix, cladothrix, yeast, sarcina, oidium albicans, jodococcus vaginalis Miller, staphylococcus pyogenes aureus and albus, spirochaeta bucallis, bacilus subtilis, and glossophyton Dessois. In some cases none or very few are to be found (Lannois,[45] Dessois,[25] Schech[85]).

The distribution of the bacteria is irregular in most cases. Some papillae may be entirely covered with microorganisms while others have only a very few, the number of bacteria having no relation to the extent and severity of the affection. These facts do not support the parasitic theory. Up to this time no one has succeeded in finding a parasite which is present in all cases of black tongue. Only a few of the reported bacteria can be considered; first, the dark moulds cultivated by Ciaglinski and Hewelke[20] (1 case); Schmiegelow[86] (2 cases); Paltauf[90] (1 case), and Sendziak[89] (2 cases). It is a striking fact that in these six cases we have to deal with six different moulds. Further, it is strange that none of the other authors has found moulds. Rydygier,[81] Vidal,[100] Lancereaux[43] and Sell[86] found mycelia, but nobody has been able to cultivate dark moulds from a black tongue, although several investigators (among others Moure[61] and Blegvad in all their cases) have used wheat-bread-gelatine, which should be the best culture medium.

It is a strange fact that the moulds mentioned can only be cultivated at 21° to 25° C., while they grow in the mouth at 37° C. Some explain this by saying that the moulds grow in mouths of patients who breathe through their mouths, where the temperature is below the normal and the tongue immobile. This explanation can only hold in those cases where the patient uses mouth respiration. In some of Schmiegelow's and Sendziak's patients this was not true. These patients were suffering with a chronic affection while with the others it was an acute affair. Thus there is not a single case reported in literature that is convincing, and one is inclined to believe that the finding of moulds in all these cases was purely accidental. Moulds are present everywhere, as is well known, and it is not probable that they should play any part in the etiology of the disease.

Another parasite which might be considered is the "spores of moulds" which Raynaud[73] has described. Dessois[25] named them glossophyton and Vignal[101] considered them identical with his alphacoccus. Many investigators have found this microbe (Blegvad in one case, Brosin,[14] Lancereaux,[48] Laveau,[46] Lohéac,[50] Masoin,[54] Pasquier,[68] Rayer,[72] Wallerand[96] (in two cases), Weill[98] and Vignal[101]). Many investigators have searched in vain (Blau,[9] Blegvad in 4 cases, Tereol[29] Mourek,[62] Richter,[76] Stocker[93] and Wallerand[96] in one case). This parasite is sometimes found on healthy tongues (Ch. Rayer,[72] Vignal[101]), and Brosin[14] is perhaps right when he says that the parasite is only a badly developed oidium albicans.

Inoculation.—It is evident from what has been said above that microscopic examinations and cultures have not given reliable support to the parasitic theory. In addition, no one has succeeded in inoculating the affection on the tongue of a man or an animal; not even in those cases where the experiments were done under the most favorable circumstances (as for instance by Wallerand[96]) have the investigations given a positive result. Wallerand tried to inoculate both papillae and cocci from a culture taken from a case of black tongue, on to the tongue of a patient with phthisis, in the last stage of the disease. Rostowzew[78] made an attempt on animals and man, Mourek[62] on man, Masoin[57] on animals, Dessois,[25] Gundobin[34] and Rayer[72] on themselves. In all cases the results were negative.

Infection.—It could be expected that black tongue, supposing it to be of bacterial origin, would sometimes be carried from one person to another by infection. Rostowzew[78] is the only author who mentions a case that could be explained in this way. He reports the case of a man and his wife who were both affected with black tongue, but this could be due just as well to the fact that both eat the same food. These facts I think prove that there is no ground for the parasitic theory.

Among the remaining theories there is only one that may be brought forward and that is the theory of Schech that black tongue is due to an hyperkeratosis of the epithelium of the filiform papillae. The proof of this theory can be gained only by microscopic examination.

It is remarkable that most of the investigators have been satisfied with making only an examination of the fresh papillae, treated with acetic acid or potassium hydroxid and staining with methylene blue, Bismark brown, or similar stains. I feel sure that the results would have been different if they had examined double-stained paraffin- or colloidin-sections. In specimens made in this way, the superficial cells are keratosed and are either without a nucleus or with a very faintly stained one. The long thin papillae consist exclusively of such cells firmly bound together. But when specimens from normal or furred tongues are examined in the same way, it is found that aside from the length of the papillae there is no essential difference between these papillae and those from a case of black tongue. I have examined microscopically curetted papillae from five normal and two furred tongues, and from one tongue (postmortem) where the tongue had been hardened by 10 per cent formalin. In all the sections numerous papillae filiformes were found, smaller than those from black tongue but otherwise not differing essentially. The superficial cells were keratosed to the same extent, had no nuclei, became orange after v. Giesen and red after Ziehl-Nielsen. The specimens differed mostly with regard to the bacteria. In some of the sections the papillae were covered with a thick layer of leptothrix (short, thick bacilli arranged in chains, Gram-positive), while in others only a few or no leptothrix were present, but large masses of zooglea consisting of bacilli and cocci. It is thus evident that the papillae from normal tongues on an average are just as much keratosed as those from black tongues and we may assume that the intense keratinization cannot be the only cause of the color in this disease. In one of the acute cases which I treated the black tongue developed in the following manner: A white spot first appeared extending from the elongated papillae filiformes and in the course of three or four days it became gray, then brown and finally black. The disease spread slowly to the different parts of the tongue so that it was possible to obtain specimens from white and black areas at the same time. I made sections of the two and found them to be the same microscopically. The keratinization, bacteria, etc., were alike except that in the unstained specimens the papillae from the dark spot were somewhat darker in color than those from the white area.

As a result of the above investigation I feel safe in saying that it is not yet proven that black tongue is due to an excessive keratinization and pigmentation of the epithelial cells of the papillae filiformes. The superficial cells of the papillae of the tongue including the papillae valatae, are always keratosed and there is no reason to assume that they should be more so in black tongue than in a normal tongue. The degeneration of the horn-color, which is mentioned by some authors, is also only hypothetic and its existence should be proven by the following facts: (1) The black color of the horn-pearls in epithelioma is due to a degeneration of the horn-color, and (2) the color in black tongue is insoluble in water. The latter fact may be due to a penetration of the epithelial cells by a color from without which cannot be washed out again, as in the staining of horn-cells with picric acid.

It is apparent that none of the foregoing theories are capable of standing a thorough criticism and that we must find a new explanation for the etiology and pathogenesis of black tongue.

In my opinion the disease originates in those persons with normally well developed papillae filiformes which have for some reason become prolonged and then have become stained by some chemical process from without. The prolongation of the papillae may be caused in different ways. It may be due to some form of irritation as smoking, to the elimination through the mouth of certain drugs as mercury, or as is oftenest the case to a slight glossitis brought about by an affection of the mouth, nose, throat or intestinal tract. The hypertrophy may be due to a nutritive disturbance or to mouth-breathing, especially in old and sick persons. Perhaps bacteria, too, may play a part. It is difficult to say whether or not black tongue is accompanied in all cases by a stomatitis as the microscopic examinations which have been made were made on papillae which were simply curetted off from the tongue. Goodale[22] says that in the corium he found an infiltration of small round cells with a protoplasmic reticulum, and I found in one case some leucocytes in the base of the papillae.

The part that the acidity of the saliva plays in the hypertrophy of the papillae is not clear. Some authors attribute great importance to this factor. In my opinion it can just as well be a matter of secondary importance due to the same cases as black tongue: immobility of the tongue, indigestion, stomatitis with fermentation and subsequent formation of acid.

The coloring of the prolonged papillae is due first of all to food (and wine); also tobacco (especially chewing) and drugs (iron, mercury, etc.). Probably mouth-breathing also has some influence since the drying of the surface of the tongue may produce a dark color.

This theory has already been partly advanced by Gallois[30] (1869) and Rydygier[81] (1891) and it is supported by several facts. The different cases of black tongue have different colors and it has been often found that different colors follow one another in the same patient at different times (black, blue, brown) (Rydygier,[81] Mickulicz[59]). Also black tongue may change to a white tongue with long, uncolored papillae.

A factor in favor of this theory is the importance of tobacco in the development of black tongue. Most authors do not mention the use of tobacco and in the cases reported by Brydon,[15] Lake,[42] Masoin[54] and Master[55] tobacco was not used at all. However, 16 of the reported cases of black tongue were in patients who smoked tobacco and of these 10 were very great smokers. In 6 of these patients no other disease was mentioned and we are justified in assuming that they were otherwise healthy.

The other 10 suffered with different diseases. Four had syphilis, 2 had angina, 1 had enteritis, and 1 colica saturnina. The importance of tobacco in the etiology of black tongue is evident in Maraval's[53] case. In a man, 42 years of age, the disease disappeared as soon as the patient abandoned the use of tobacco, but reappeared with all the characteristic symptoms when the patient, two months later again began the use of the tobacco. Among the 73 adults with black tongue, there were 16, or 22 per cent who were smokers, and 30 per cent of the 45 men also smoked tobacco. These figures are probably too small as few authors mention the use of or abstinence from tobacco in their patients.

The influence of drugs is to be noted in the following cases: Dinkler,[26] in case No. 3, reports a patient who had white tongue which appeared and disappeared and finally developed into a black tongue when the patient took iron-powder. The scraped-off papillae gave a reaction with ferrocyanid of potassium and included a granulated pigment. Blair[9] (case No. 4): woman, 21 years of age, with sinusitis maxillaris operated through the alveolus; first, grayish-yellow hairs were seen on the tongue and two days later these hairs were colored black through the use of iodoform gauze. They disappeared spontaneously. Balfour-Graham[5] reports a case of black tongue, in a patient suffering with caries of the first dorsal vertebra, which developed while he was taking large doses of mercury. It seems that at the same time he had a severe stomatitis, for all of his teeth fell out.

Cases are also reported where the disease has developed during the use of opium (Hofheimer[35]) and bismuth (one of our cases). It can be easily understood that black tongue may develop after the application on the surface of the tongue of chromic acid (Leviseur,[49] two of our cases) or silver nitrate (Schmiegelow[86]). The importance of coloring matter from without is evident from the following case, cured by us. The patient, a man, 21 years of age, was suffering from a violent attack of mercurial stomatitis during which he developed a case of black tongue. The black color was seen not only on the tongue but also on the teeth which were entirely covered with a thick, shiny, black coating. This coating was scraped off and examined chemically. It consisted of organic substances, potassium and sodium but did not contain any mercury. This goes to prove that mercury does not act in this way, since black mercuric salts (viz., mercuric sulphide) are found in the mouth. It is probable that the mercury by its excretion irritates and thus produces a stomatitis on the basis of which the black tongue develops. When black tongue is so often found in persons suffering from syphilis it is probably because syphilis is so frequently accompanied by affections of the mouth.

There is, in my opinion, no difference between a black tongue and the ordinary furred tongue. Müller[109] and Fuchs[108] emphasize the fact that there are two different kinds of furred tongues. The genuine fur consists of loosened epithelium, leucocytes, moulds, residues of food, etc. The "not genuine" fur is made up of the hair-like prolonged papillae filiformes with their keratosed epithelial cells. Naturally it is the latter kind of furred tongue that the black tongue resembles.

The location of the black spot just in front of the papillae vallatae is explained by the fact that the papillae filiformes normally are most developed at this place (Koelliker[106]) and that the friction against the teeth and palate is less here.

Black tongue sometimes has a very acute course and occasionally lasts for years. This will be understood when we bear in mind that the etiologic factors (the nutritive irritation and the coloring matter) sometimes are present for a long time and sometimes for only a very short time.

The fact that the color has its seat especially in the uppermost parts of the papillae depends upon the fact that the food in passing as well as the drugs, etc., only touch the tips of the papillae.

DIAGNOSIS.

In making the diagnosis lingua villosa nigra, we need only consider the two principal symptoms: (1) The hypertrophy of the papillae filiformes and (2) the dark color. Not every dark stained tongue, however, can be called a black tongue. For instance, the normal tongue may be temporarily colored by certain coloring matters passing through the mouth. The tongue is colored black by ink, red wine, mulberries, and certain kinds of cherries; it is colored brown by licorice, fresh nuts and dried plums, and brownish-gray by chocolate. Tannin preceded by iron also stains the tongue black. Black tongue cannot easily be confounded with leucoplakia oris (psoriasis linguae) because in the latter the spots of thickened epithelium are either white or gray, but in persons who smoke tobacco to excess, and such persons are more likely to be affected with leucoplakia, the epithelial hypertrophy may be grayish-black. The surface of the spots is perfectly smooth and the papillae do not show any hypertrophy. Moreover, the spots most commonly are found on the borders and the tip of the tongue and inside the cheeks. Accompanying the morbus Addisonii, one often finds in the mouth the so-called melanoplakia, dark spots due to the deposit of pigment in the rete malpighi, without proliferation of the epithelium. These spots, however, appear mostly on the lips and inside the cheeks. Patients who suffer with tuberculosis of the lungs often show signs of melanosis, especially in the face. The spots begin to make their appearance on the nose or forehead and seldom spread to the mucous membrane. If this does occur the affection must be distinguished from lingua villosa nigra which also, as before stated, ocassionally is found in tuberculosis patients.

TREATMENT.

Since black tongue is not dangerous to life and since it very often disappears spontaneously, it needs treatment only in cases where it is accompanied by disagreeable symptoms, viz., pain, disturbance of taste or the like, or when a neurasthenic or hypochondriac patient asks for treatment.

In times past, a great many different remedies have been proposed and used, a fact which shows that the affection is resistant to most of them. A therapeutic procedure used in very many cases is to scrape off the papillae with a curette or to cut them off with a pair of scissors. This treatment nearly always causes the affection to recur in a few days, since it acts as an irritant which stimulates the growth of the papillae. The use of cauteries such as lactic acid, chromic acid, silver nitrate, trichloracetic acid, etc., shows a similar result, because of which they are unsuitable for treatment. They increase the existing irritation and besides in many cases increase the dark color.

When we consider that black tongue has been thought to be of parasitic origin, it is understood that almost all of the known antiparasitic remedies have been used but always without producing any considerable effect (bichorid of mercury, carbolic acid, boric acid, etc.).

In support of the theory that black tongue is an hyperkeratosis there have been applied horn-dissolving remedies (salicylic acid) but this treatment has also been unsatisfactory, an indirect proof for the untenableness of the hyperkeratosis-theory.

The best remedy is the local application of a 10 per cent solution of hydrogen peroxyd which destroys directly the coloring matter by the influence of its oxygen (which acts in statu nascendi). The remedy must be applied every day and sometimes where the color is very dark two applications daily will be necessary.

As the cause of black tongue is always a slight glossitis or stomatitis some form of mouth wash should be used (chlorate of potassium, boric acid, permanganate of potassium).

The patient should always be told that the affection is very apt to recur, especially when the causes of the affection (use of tobacco, indigestion) are not removed.

BIBLIOGRAPHY.

A. Publications directly concerning Black Tongue.

1. Abercrombie. A case of Black Tongue. British Laryngol., Rhinol. and Otol. Ass'n, Nov. 8, 1901, Centralbl. f. Laryng., Vol. 19, 1903 p. 214.
2. Armaingand. Revue des sciences medicales, Vol. xi, p. 185.
3. Anderson Smith. Black Tongue. Brit. Med. Jour., 1891, p. 946.
4. Balfour, Graham R. Black Tongue. Brit. Med. Jour., 1887, Vol. i, p. 61.
6. Barnes. Black Tongue. Brit. Med. Jour., 1884, Vol. i, p. 995.
7. Bernhardt. Zur schwarzen Haarzunge. Mon. für prakt. Dermatol., No. 8, 1888; Centralbl. f. Laryngol., 1889, Vol. 5, p. 108.
8. Birch-Hirschfeld. Lehrb. d. patholog. Anatomie., Leipzig, 1887, p. 468.
9. Blau, Albert. Die Haarzunge. Archiv. f. Laryng., Bd. 15, 1904, Heft. 2, p. 263.
10. Boston Med. and Surg. Jour., 1835, iv, pp. 65-78. Blackness of the Tongue.
11. Boucher. Thèse de Lille, 1903, cit. Papon, Arch. de med. et de pharm. milit., Dec., 1903, p. 541.
12. Priz. Un Notable Cas de Lingua Nigra. Rev. de Terap. y. Farm., Madrid, 1891, Vol. 2, p. 105; cit. Johnson, N. Y. Med. Journ., 1893, p. 126.
13. Broatch. Case of Black Tongue. Brit. Med. Journ., 1884, Vol. i, p. 761.
14. Brosin. Ueber schwarze Zunge. Dermatol. Studien., Heft. vii, 1888.
15. Brydon, James. Black Tongue. Brit. Med. Journ., 1884, Vol. i, p. 761.
16. Bocher, C. Lingua Nigra. Hospitals Tidende, 1887, p. 221.
17. Butlin, H. Krankheiten der Zunge. German Translation by J. Beregszaszy, Wien, 1887, p. 26.
18. Butlin and Spencer. Diseases of the Tongue. London, 1900, p. 145.
19. Chevalier. De la langue noire. Archiv. de la med., Belges, November, 1904.
20. Ciaglinski and Hewelke. Ueber die sogenannte schwarze Zunge. Zeitschrift f. klin. Med., Bd. xxii, 1893, p. 626.
21. Cohen, Solis. Two specimens of Melanotic Hypertrophy of the Filiform Papillae of the Tongue. Trans. of the Path. Soc. of Philadelphia, 1885, pp. 7, 13, 70.
22. Cornil and Ranvier. Manual d'histologie pathologique.

23. Curtis. A Painful Fungus Growth of Unknown Character at the Base of the Tongue. Proceedings of the N. Y. Academy of Med., April 23, 1889, N. Y. Med. Journ., 1889, Vol. 2, p. 216.
24. David. Les microbes de la bouche, 1890. Langue noire, p. 164.
25. Dessois. De la langue noire. Thèse Paris, 1878.
26. Dinkler. Ein Beitrag zur Pathologie der sogenannten schwarzen Haarzunge und einer ihr verwandten Form der Zungenschleim hauterkrankungen. Virchow's Archiv. Bd. 118, p. 46,. 1889.
27. Eulenberg. Ein schwarzen Zungenbelag. Archiv. f. physiol. Heilkunde, 1853, Vol. xvii, p. 490.
28. Eve, T. Black Tongue. Trans. of the Clinical Soc. of London, 1893-94, Vol. xxvii, p. 277.
29. Feréol. Note sur un cas de coloration noire de la langue. Société méd. des hôpitaux, June 25, 1875; L'union medicale, Sept. 14, 1875, p. 393.
30. Gallois. Sur un cas de coloration noire de langue. Compt. rend. de la soc. de biol., Paris, 1869.
31. Gaston et Nicolau: Société francaise de Dermatologie et de Syphilographie, Feb. 5, 1903.
32. Goodale. A contribution to the Pathological Histology of Hyperkeratosis lingualis. Annals of Otology, Rhinology and Laryngology, Feb., 1900. Centralbl. f. Laryng., etc., Vol. xvii, 1901, p. 83.
33. Gubler. Bouche (semeiologie). Diction. encyclopedique des sciences medicales, December, Vol. x, p. 230.
34. Gundobin. K aetiologiu tschernawo jasika u detei (Etiology of Black Tongue in Children). Med. Oborzeme, 1888, No. 19, Centralbl. f. Laryng., etc., 1890, Vol. vi, p. 19.
35. Hofheimer. A contribution to the Study of Nigrities. N. Y. Med. Record, 1890, Vol. xxxviii, p. 698.
36. Horand et Weil. Soc. des sciences med. de Lyon, Oct. 31, 1888.
37. Hutchinson. Med. Press and Circular, Vol. ii, 1883, p. 20.
38. Johnston. A Case of Black Hairy Tongue. N. Y. Med. Jour., 1903, p. 126.
39. Johnston, R. H. A Case of So-called Hairy Tongue. The Laryngoscope, July, 1905.
40. Krimer. Merkwürdiger Fall von Schwarzwerden der Zunge ohne wahrnehmbare materielle Ursache. Med. Konversationsb., 1830, Vol. i, pp. 73-76.
41. Lack. Cyste der Epiglottis und schwarze Zunge. Brit. Laryng., Rhin. and Otol. Ass'n, March 10, 1897; Centrlb. f. Larnyg., etc., Vol. xviii, 1897, p. 530.
42. Lake, R. Black Tongue. Brit. Med. Jour., 1891, Vol. ii, p. 946.
43. Lanceraux. Note sur un cas de langue noire. Soc. Med. de hôpitaux, Dec. 8, 1876; L'Union medicale, 1877, Vol. xxviii. p. 459.
44. Landouzy. La "langue noire," sa premiere description par H. Landouzy en 1845. Union medicale du Nord-Est., Sept., 1845; cit. Monats. f. Ohrenheilk., 1897, p. 83.
45. Lannois. Sur la langue noire. Ann. des mal. de l'oreille, etc., 1888, p. 568.
46. Laveau. De la langue noire. Thése, Paris, 1876.

47. Lecocg. Langue noire. Soc. Anatomo-clinique de Lille., March 10, 1897.

48. Lediard. Black Tongue (card specimen). Trans. of the Path. Soc. of London, Vol. xxxvii, 1885-86, p. 222.

49. Leviseur. Black Tongue. N. Y. Med. Jour., Vol. 49, 1889, p. 42.

50. Loheac. Two Cases of Black Tongue. Journ. des sciences med. de Lille, May 1, 1897; N. Y. Med. Journ., 1897, p. 714.

51. Lusitanus, Amatus, cit. Arnold. Virchow's Arch., Bd. iii, p. 888.

52. Mahe. Note sur un cas de langue noire. Gaz. med. d'Orient, Constantinople, 1881, p. 10.

53. Maraval. Contribution a l'etude de la melanotrichie linguale (observation). Revue hebdomadaire de Laryngologie, Vol. xx, 1900, p. 273.

54. Masoni, P. De la langue noire. Bull. de l'academie royale de med. de Belge., iv Serie, Vol. vii, 1893, p. 387.

55. Masters, J. B. Black Tongue. Brit. Med. Journ., 1891, Vol. ii, p. 1043.

56. Mathieu. Bull. de la soc. anatom., 1882, p. 535.

57. Melchior, R. P. Observation nouvelle de la langue noire. Marseille med., Dec. 18, 1897.

58. Mikulicz und Michelson. Schwarze Zunge. Atlas der Krankheiten der Mund- und Rachenhöhle, Berlin, 1892, Vol. ii, Tafel xxxv, Fig. 1.

60. Montgomery. The Black, Hairy Tongue. Pacific Med. Journ., 1892, Vol. 35, p. 4.

61. Moure. Mem. de la soc. de med. et de chir. de Bordeaux, 1883, pp. 437-602.

62. Mourek, H. Ueber de grüne Haarzunge. Archiv. f. Derm. und Syph., Bd. 29, 1894, p. 369.

63. Mracek. Die Erkrankungen der Mundhöhle. Nothnagels spez. Path. u. Ther., Bd. 16, Teil 1. Abt. 1, p. 349.

64. Norsk Magasin for Laegevidenskaben, 1885, p. 97.

65. Pallarez. Ampiteatro anatomico espagnol, 1879.

66. Papon. Un cas de langue villeuse rapidement guerie par l'eau oxygenée. Arc. de med. et de pharm. milit., December, 1903, p. 541.

67. Parmentier. La langue noire. Le Progres med. Belg., Vol. xxiv, 1900.

68. Pasquier. Deux cas de glossophytie. Bull. Med. du Nord., May, 1883.

69. Potter, Furniss. Black Tongue. Brit. Laryng., Rhin. and Otol. Ass'n, Centralb. f. Laryng., etc., Vol. xvi, 1895, p. 328.

70. Ramsey. Singular Disease of the Tongue. South. Journ. of Med. and Phar., Charleston, 1847, Vol. ii, p. 533.

71. Rayer. Nigritie. Traite des mal. de la peau., 2nd edit., iii, 1835, p. 573.

72. Rayer, Ch. Des langues noires. Thèse, Paris, 1883.

73. Raynaud, Maurice. Note sur une nouvelle affection parasitaire de la mucueuse linguale. Compt. med. de la soc. med. des hôpitaux, January 26, 1869; L'Union med., 1869, pp. 3-14.

74. Raynaud, Maurice. Comp. rend. de la soc. des hôpitaux, June 25, 1875.

75. Read. Black Tongue. Boston Med. and Surg. Journ., 1860, lxii, p. 21.

76. Richter. Die neuen Kentnisse von den Krankmachenden Schmerotzerpilzen. Schmidt's Jahrbücher, 1871, Bd. 151-152, p. 313.

77. Rosbach. Ueber schwarze Zunge. Gesam. klin. Arbeit., June, 1887.

78. Rostowzew. Ueber die schwarze Haarzunge. Bolnit. Gas. Botkmies, 1896; Ergebnisse der allgem. Path. und path. Anat. (Lubarsch u. Ostertag), v, 1898, p. 731.

79. Roth. Ueber haarförmige Bildungen an der Zunge. Wien. med. Presse, 1887, 26, 27, p. 897.

80. Rumsey. Remarkable Blackness of the Tongue. London Med. Gazette, 1830-31, Vol. vii, p. 245.

81. Rydygier, L. Beitrag zu selteneren Erkank. der Zunge. Archiv. für klin. Chir., Bd. 42, 1891, p. 767.

82. Salter, Hyde. Todd's Cyclopedia of Anat. and Phys., 1852, Vol. iv, p. 1159.

83. Schech. Die Krankheiten der Mundhöhle, des Rachen und der Nase. Wien, 1885, p. 36.

84. Schech. Die Krankheiten der Mundhöhle, des Rachens, etc. Wien, 1896, p. 24.

85. Schech. Die "schwarze Zunge." Münchner med. Woch., Vol. 34, 1887, p. 253.

86. Schmiegelow. Beitrag zur Pathogenese der sogenanten schwarzen Zunge. Arch. f. Laryn., etc., Bd. 4, Hf. 2, 1896.

87a. Sell. Tilfaelde af "Lingua nigra." Hospitalstidende, 1879, p. 977.

87b. Sell. Tilfaelde af "Lingua nigra." Hospitalstidende, 1885, p. 87.

88. Semon. Black Tongue. London Laryn. Gesell., Jan. 13, 1897; Centr. f. Laryn., etc., Vol. 13, 1897, p. 449.

89. Sendziak. Contribution a l'etiologie de la soi-disante langue noire. Communication facté à la soc. polonaise de Lar., Rhin. et Otol., Varsovie, November, 1893; Revue mensuelle de Laryn., etc., Bd. 14, 1894, p. 228.

90. Sendziak. The Etiology and Treatment of Mycosis Occurring in the Upper Respiratory Tract. The Journ. of Laryn., Rhin. and Otol., Vol. xx, 1905, p. 567.

91. Saint-Germain, Bertrand de. Nigritie de la langue en dehors de tout etat febrile. Compt. rend. des sceances l'academie des sciences, Paris, 1885, Vol. 41, p. 932.

92. Stokes, J. R. Black Tongue. Brit. Med. Journ., 1886, Vol. ii, p. 629.

93. Stokes, G. Black Tongue. Brit. Med. Journ., 1884, Vol. i, p. 661.

94. Stokes, G. The Brit. Laryn. and Rhin. Ass'n., June 13, 1890; Centr. f. Laryng., etc., Vol. vii, 1891, p. 401.

95. Surmount. La langue noire, melanotridrie linguale. Gaz. des hôpitaux, July 6, 1890.

96. Wallerand. Contribution a l'etude de l'etiologie et de la path., de la langue noire pileuse. Thèse, Paris, 1890.

97. Vallin. Cit. Wallerand.[96]

98. Weill, P. E. Soc. de Derm. et de Syph., November, 1903; cit. Centralb. f. Laryng., etc., Vol. 20, 1904. p. 187.

99. Vermet. Un cas de nigritie de la langue. Gaz. des hôpitaux, Paris, 1887, 69, p. 1346.

100. Vidal. Soc. med. des hôpitaux, Dec. 8, 1876.
101. Vignal. Recherches sur les micro-organismes de la bouche., Archiv. de Phys., Vol. viii, 1886, p. 350.
102. Villar. Langue noire. Journ. de med. de Bordeaux, April 11, 1895.
103. Vollmer, E. Histologische Bemerkungen zu einen Fall von schwarze Haarzunge., Archiv. f. Dermat. und Syph., Bd. 46, 1896, p. 13.

B. Publications that do not directly concern Black Tongue.

104. Bernabei. Sul fundamento di una diagnostica profilathica dell' infezione d'origine bocalle e sulle ceiuse delle autointossicazione che procedone dell' apparato digerente. Estratto dal Bolli. della R. acad. med. di Roma, cit. Centralb. f. innere Med., 1894, p. 864.
105. Fuchs. Ueber die Zungenbelag und seine Bedeutung nebst Untersuchungen über einige chemische Vorgänge in der Mundhöhle nach der Nahrungsaufnahme, Dissert., Würzburg, 1898.
106. Koelliker. Handbuch der Gewebelehre des Menschen., Bd. 3, 4, Victor v. Ebner, Leipzig, 1902.
107. Lange, C. Fordijelsesorganernes patologiske Anatomi oz Patologi, Kobenhavn, 1893.
108. Mills. Die Mikro-organismen der Mundhöhle, Leipzig, 1889.
109. Müller, J. Ueber den Zungenbelag bei Gesunden und Kranken. Münchener med. Woch., xlvii, 1900, p. 1125.
110. Unna. Monatshefte f. prakt. Dermatologie, Bd. 4, 1885, No. 9, and Dermatologische Studien, Heft. vii, 1888.

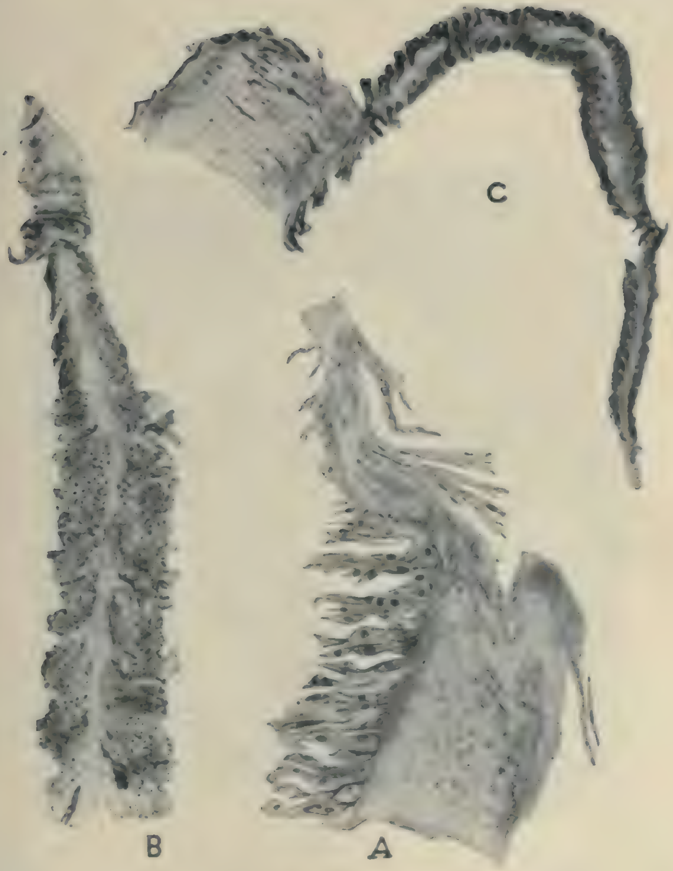

FIGURE 1.

A.—Black Tongue I. Paraffin slide of papilla filiformis. Van Giesen stain. Enlarged 220 times. Tile-shaped epithelial cell on surface.

B.—Black Tongue II. Paraffin slide of papilla filiformis. Van Giesen stain. Enlarged 220 times. Numerous large cocci (Spores(?) Dessois glossophyton) on the surface.

C.—Same specimen as B., 70 times enlarged, showing two papillae connected with epithelial cells at their base.

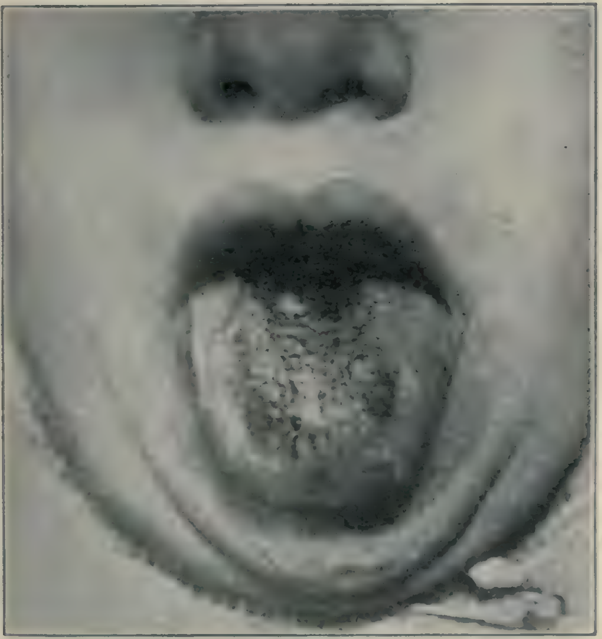

FIGURE 2.

Black Tongue I. Caroline F., aet. 22. Syphilis and severe mercurial stomatitis. After a local application of chromic acid, developed black tongue which lasted for more than a month with intermissions.

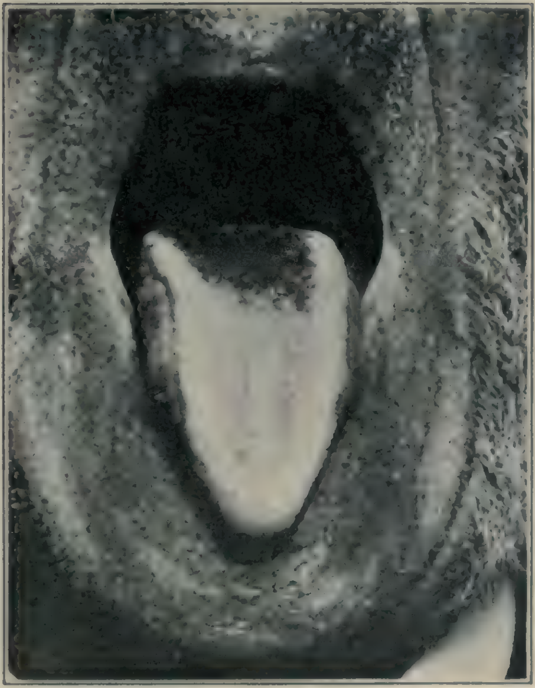

FIGURE 3.

Black Tongue II. Christian C., aet. 70. Progressive bulbar paralysis and a chronic intermittent black tongue.

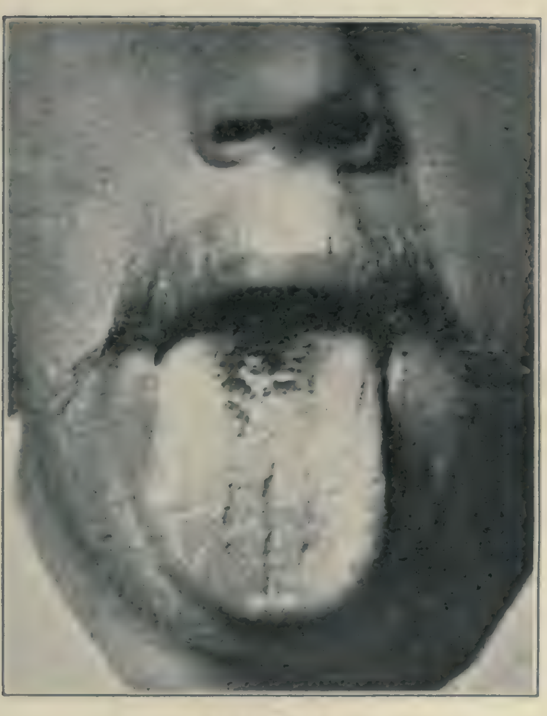

FIGURE 4.

Peter C., aet 23. Syphilis with no stomatitis. Acute case of black tongue.

XXXII.

PAPILLOMA OF THE LARYNX IN CHILDREN; A FURTHER CONSIDERATION OF THE SUBJECT.*

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Three years ago I had the privilege of reading a paper on this subject before this Association. I wish now to report the results in the four cases which were then incomplete, to report four new cases and to review the literature which has appeared in the past three years, and, in the light of more experience and thought, to modify somewhat the statement which I then made in regard to its treatment.

In reporting the subsequent histories of cases 9, 10, 13 and 14 in my first paper, I will begin with a brief resumé of each case as they were then reported.

Case 9.—Boy, 6 years old, when first seen in February, 1901, with symptoms of a year's duration. Tracheotomy for severe dyspnea. Papilloma removed. Eight months later, larynx again filled with growth. Papilloma again removed. In February, 1902, larynx free from growth above vocal cords but large mass below glottis. Nothing done. Seen from time to time, growth diminishing. In October, 1904, only a small growth below glottis, growing from anterior wall. This was the condition of the case when last reported. On June 27, 1905, he was admitted to the hospital and, under ether anesthesia, a small growth was removed from the larynx through the Killian tube spatula and a small mass from the tracheal wall through the tracheal opening. On July 5th, no papilloma was visible in larynx or trachea. September 11th, no papilloma. Tube removed. The patient reported again on March 17, 1906, when the larynx was normal. Under ether anesthesia the fistula left by the tracheotomy tube was dissected out and the wound sewed up. Recovery was uneventful.

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*Read at the Congress of the American Laryngological Association in Montreal, May, 1908.

Case 10.—George W., 9 years old, when first seen in August, 1904, with history of hoarseness for six months and dyspnea for two weeks. Tracheotomy performed. Growth removed in September, 1904, and again in February, 1905. It recurred rapidly and further operative interference was abandoned. Such was the state of the case when I reported it. On May 15, 1905, the record says: Papilloma not increased in size since last visit. Rises from right side and anterior commissure. July 10th: Papilloma appears smaller. August 28th: Growth about the size of half a split pea in anterior commissure. December, 1905: The same. May 11, 1906: Papilloma having remained quiescent for ten months it was decided to remove what remains. The patient was etherized and placed in the Rose position. By means of the tube-spatula a good view of the larynx was obtained. A growth the size of a pea was seen on the anterior tracheal wall below the vocal cords and was removed with alligator forceps and curette. On May 24th, there being no evidence of papilloma anywhere, the tracheotomy tube was removed. It had been worn for one year and nine months. On July 18, 1906, the edges of the tracheal fistula were refreshed by dissecting out the scar tissue and the edges brought together and sutured. July 22, 1907: Larynx free from papilloma, vocal cords normal in appearance, voice good. Tracheotomy wound has healed nicely.

Case 13.—Nellie S., aged 6, entered the hospital on July 5, 1904, with a large papilloma, completely hiding the glottis. Tracheotomy was done and on September 26th the growth was removed. It recurred rapidly and over a greater area than before the operation. Under these conditions non-interference was decided on. Note on May 5, 1905: Large papilloma visible through tracheotomy wound, apparently attached above. No change in larynx. September 22nd: Comes in with history of having been ill with pneumonia since June 28th. Papilloma is much diminished. None visible through tracheotomy wound. Breathes fairly well with tube closed and can speak aloud. Examination of chest shows dullness and diminished breathing over lower half of right side, front and back, slight dullness at left base behind. X-ray showed opaque shadow from fifth space to base of right lung with less dense shadow at base of left lung. It was thought that there was fluid on right side but a needle was inserted with negative results. The patient's general condition rapidly improved in the hospital. Her temperature became normal on October 4th and she felt perfectly well. She was discharged relieved on October 12th.

Diagnosis.—Thickened pleura and chronic bronchitis.

On November 10th the papilloma had increased somewhat, and her general condition was poor. Her temperature was 100, pulse 160 and respiration 40.. Physical examination of the chest gave practically the same condition as in October. On November 24th, the condition on the right was about as before, but in the left axillary line there was a dull area about 2½ inches in diameter with fine moist rales. Broncho-pneumonia. The patient had a temperature of 102, chilly feelings, poor appetite, pain in back and sides on cough, which was frequent. The temperature dropped to normal on night of the 24th and remained so. The sputum at this time was large in amount, containing a few influenza bacilli, many pneumococci, some in long chains, all extracellular, and many other bacteria but no bacilli of tuberculosis. On November 29th she was discharged "relieved." The diagnosis was croupous pneumonia. She died at home on February 12, 1906. A letter from the family physician, dated June 16, 1906, contains the following: "The condition of Nellie S. during last few weeks was one of exhaustion and pyemia. She took little nourishment, vomited more or less and raised through tube large quantities of fetid pus, as much as four ounces at a coughing spell. She complained of some pain in region of heart during cough. She had not been able to lie down for a number of months. Sputum showed no tubercle bacilli after repeated examinations during last few months although tuberculosis is in her immediate family."

CASE 14.—Jennie M. C., age 2 years, was admitted August, 1904, on account of increasing hoarseness and dyspnea. An examination showed the larynx almost filled with papilloma. Tracheotomy was done and in October the growth was thoroughly removed. In February, 1905, the larynx was again full and it was decided not to attempt further removal. On September 8th papilloma was seen presenting at the tracheal opening as well as in the larynx. On January 19, 1906, while changing the tube, the child was nearly asphyxiated by the closing of the tracheal opening by a papilloma. A portion of this growth was removed. On March 1st a small papilloma, springing from the tracheal wall was removed through the tracheal opening. On May 21st the growth had diminished. On July 9th the remaining growth was removed through a Kierstein autoscope.

February 13, 1907.—Very small papilloma, 1 to 2 mm. in diameter on anterior portion of left vocal cord.

February 18th.—A small papilloma removed with snare through tracheal opening.

April 10th.—Growth on cord nearly disappeared. No recurrence in trachea. Tracheotomy tube left out.

September 25th.—Growth has gone. Left vocal cord slightly reddened at site of growth.

In April, 1908, the larynx being normal in appearance the patient was admitted to the hospital, the tracheal fistula dissected out and the wound sutured.

Since the above cases were first reported there have been four other cases in the Massachusetts Hospital throat clinic which I will report:

CASE 15.—Alphocena M., girl, age 6, was first seen on January 10th, 1905. She had convulsions a year before and lost her speech a few days after. Speaks now only in a whisper. The larynx appeared to be two-thirds full of papilloma. On January 14th a small piece was removed. On February 11th the growth was seen to have an extensive base but no large pieces. There is no record after this and a letter written to the parents on March 11, 1908, was returned stamped by the post office authorities, "Removed, address unknown."

CASE 16.—Herbert H., age 10, came to the clinic on March 14, 1905, with the history of hoarseness and slight cough for a year. On examination a papilloma was seen apparently protruding from the left ventricle and a smaller one in the anterior commissure. On March 21st the Hooper laryngeal curette was passed twice but no growth obtained. On April 13th there was no material change in the size of the papilloma. On May 25th the growth had nearly disappeared and the voice was fairly loud. The patient was written to in March, 1908, and the following reply received: "The boy's throat has been very well since the last visit. He has grown rapidly and Dr. B., our local doctor, thinks he has outgrown the trouble."

CASE 17.—Annie F., age 7, was seen in August, 1906. There was a history of mouth-breathing, choking at night and inspiratory stridor. The patient was aphonic and there was marked supraclavicular retraction on inspiration which was whistling. An examination showed a large papilloma on the right cord, causing marked obstruction. Tracheotomy was advised. The patient did not return and a letter sent on March 11, 1908, to the address given was returned stamped "unclaimed."

CASE 18.—Charles B., age 3, was seen first on Mary 22, 1907, with the history of having lost his voice eight months before. The laryngeal obstruction was sufficient to cause aphonia and dyspnea. Examination showed a large mass growing from both sides of the larynx. On May 24th I performed tracheotomy and the patient was discharged on the 31st, wearing the tube. After over three months of waiting, the papilloma remaining apparently the same size, the patient was etherized on September 6th and as much as possible of the growth removed with alligator forceps by direct laryngoscopy. On September 30th an examination under ether anesthesia with the tube spatula showed a very small growth remaining on the right side of the larynx. An examination on March 23, 1908, in the same manner, showed the larynx absolutely free from papilloma. The tracheotomy tube was removed and the tracheal fistula will be closed after waiting two or three months to be sure that there is no recurrence.

The outcome in case 14 adds one more death in the group first reported, making five fatal cases out of the ten in whom tracheotomy was done. The fatal termination in this case does not alter my belief that the method pursued was the right one. If the growth had been removed at first without preliminary tracheotomy and there had been no occurrence at the time requiring an emergency tracheotomy, the growth recurred so rapidly and extensively that tracheotomy would have eventually been necessary. If the growth had been removed by laryngo-fissure there is no reason to believe, from the history of similar cases, that there would not have been an equally rapid recurrence. It is obvious, however, that children wearing tracheotomy tubes should have good home care. The parents should be instructed in the care of the tube and in reasonable precautions against catching cold and that measles should be guarded against as particularly dangerous.

It hardly seems necessary to speak of the advantages of a preliminary over an emergency tracheotomy. In the former the site of the incision is carefully chosen and each step of the operation is done in a thoroughly surgical manner, while in the latter the importance of opening the trachea as soon as possible makes a careful dissection impossible. John Rogers has said something about tracheotomy which I shall quote later.

Cases 15 and 16 illustrate the type of case which gets well without tracheotomy after one removal of the growth. In these cases the papilloma was evidently in a retrogressive condition. In fact case 16 may be said to have recovered without treatment, since the one attempt to remove the growth failed. Although case 15 is incomplete it seems fair to assume from the diminution in the growth in the month between the first and last examination that this papilloma disappeared without further treatment in the same way that those in case 16 did. A bad prognosis in case 17 without treatment seems perfectly reasonable. Case 18 illustrates well a successful result of tracheotomy and the removal of the growth later when it had ceased to increase.

The literature of this subject during the past three years contains some interesting and valuable articles. A. Neubauer reports five cases on which he operated, three after a preliminary tracheotomy and two without. Four of these cases were cured and one died. This case, a girl of two years of age, had treacheotomy for dyspnea. The papilloma was removed two weeks later. After five weeks the child had measles followed by bronchitis and bronchopneumonia to which she succumbed. One case, a girl of 3 years, had had laryngo-fissure a year before, followed in six months by hoarseness and dyspnea, caused by a recurrence of the growth. This patient wore the tube for over two and a half years. The other tracheotomized patient wore the tube a year and a half. A boy, age not mentioned, had a papilloma on the right vocal cord, causing aphonia. This was removed without tracheotomy and three months later the larynx was still free from growth. A girl of three, with hoarseness for a year and dyspnea for two weeks had a papilloma on the left vocal cord, size of a bean. This was removed without tracheotomy and the patient discharged in two weeks relieved. Neither of these cases can be considered complete as in neither has sufficient time elapsed to warrant assurance of non-recurrence. Neubauer removed the papilloma in all these cases with the modified Löri catheter, an instrument with which it would seem to be impossible to effect a thorough removal of the growth, except by a happy chance.

Koelreutter reports a case in which he thinks the administration of iodid of potash in small doses caused the disappearance of the papilloma when it had recurred once after removal. It does not seem reasonable to draw any conclusions from one case when we know that papilloma sometimes disappears without treatment.

Garel reports two cases operated on through Killian's tube spatula without previous tracheotomy. Both cases exhibited marked dyspnea and tracheotomy instruments were ready. One case was operated on four times in eighteen days and the child discharged five days after the last operation with the larynx free. In the other case there was an attack of suffocation the night after the first operation for which intubation was done. This child was operated on three times in nine days then operative interference was interrupted on account of an attack of rheumatic fever. Neither of these cases are of value statistically because reported too early.

McCreary reported the case of a girl 2½ years old in which intubation gave no relief, in fact the condition was worse. Tracheotomy was done and the child is still wearing the tube.

In discussing this case John Rogers said that a high tracheotomy with a long curved tube was very liable to make a "spur" behind or a stricture if the opening was made to one side. If the wound is medial and not too high the chance of stricture is slight. Everything depends on where the incision is made. This is a strong argument against an emergency tracheotomy. In not one of my cases was there the slightest evidence of a tracheal stricture. Rogers reported that in one case he had opened the larynx and trachea and cut through the hyoid bone, excising all the papillomata and then put in an intubation tube and sewed the wound up tight. He does not mention how long the intubation tube was kept in or the condition of the voice afterwards. This operation had been done nearly a year before and there had been no recurrence. He expressed the opinion that recurrence was probably due to deficiency of excision. It is surprising that he could make such a statement as this on the strength of one case in the light of the literature of this subject.

Logan Turner showed before the Laryngological Society of London the larynx of a boy, aged 10, who died suddenly while eating, apparently choking to death. He had hoarseness but no history of dyspnea or choking before. Almost the whole larynx above the glottis was filled with papilloma.

Wingrave is inclined to consider laryngeal irritation due to mouth-breathing caused by the presence of adenoids, a factor in pathogeny. He states that papillomata have been reported not only as co-existing with adenoids but as having spontaneously disappeared after the removal of the latter. The writer is obliged to disagree with this statement. While he has had cases in which adenoids were present and were removed before the papilloma was touched, it has never been his good fortune to see any diminution of the papilloma following this operation or to find any such cases reported in literature.

Baumgarten has published an interesting and instructive article which I shall quote rather freely because his conclusions coincide closely with my statements made three years ago and with my present position on the subject. He propounds an interesting theory as to the etiology of papilloma in adults which I will not go into as it is not germane to the subject of this paper. He suggests the possibility of a gonorrheal discharge from the mother at birth being a factor in etiology. As another factor he mentions bad hygienic surroundings. He also states that blonds are more susceptible than brunettes and that adenoids stand in no causative relation. There comes a time, he says, with every child who has papilloma when the power of the papilloma to recur after operation ceases. In rare cases it ceases after the first operation. In most cases, on the other hand, it loses this power only after months or years and it is immaterial whether one operates endolaryngeally or by laryngo-fissure and removes most carefully the minutest growths and destroys the base. He says further that all cases that have measles die of pneumonia. It is my own opinion that, while measles is a very grave complication, it is not necessarily fatal. Case 4, in my series, recovered from pneumonia following measles. Baumgarten keeps the tube in for six months after the larynx is free. He advises tracheotomy for children under 10 years of age even when dyspnea is slight, recognizing the disadvantages of an emergency tracheotomy. After tracheotomy one can operate unhampered by any thought of any interference with the respiration. Children who are tracheotomized bear the operation better. Hygienic surroundings, nutrition and age affect the prognosis. In children under 6 years, if symptoms of suffocation are present or come on during examination, tracheotomy is done.

A case reported by B. Robinson illustrates the evil of not leaving the tracheotomy tube in until one can be reasonably sure that there will be no recurrence. A girl of 5 years had tracheotomy on account of cessation of respiration during examination on March 19, 1904. The papilloma was removed in two operations and the tube removed a week after the second operation. The patient returned on May 14th with severe dyspnea for which she had to be intubed. The growths were removed intralaryngeally in July. On August 27th the tracheotomy wound had to be reopened and the tube inserted. In September thyrotomy was done, the growths removed with scissors and the base cauterized. The tracheotomy tube was removed in five days. In January, 1905, the papilloma had recurred and was partially removed intralaryngeally. In February laryngotomy was again done for suffocation. The tube was removed in a few weeks. The patient was admitted to the hospital in June with bronchopneumonia. She recovered from this disease but the larynx was found to again contain papillomata. These were removed intralaryngeally from time to time. An intubation tube was occasionally inserted for dyspnea. During 1906 the growths were removed nine times, the last being in November. Breathing became bad in December and she died suddenly on January 2, 1907, of suffocation before relief could be given, nearly three years after the case was first seen. One cannot help feeling that if the tracheotomy tube had been left in uninterruptedly and no attempt at removal made until the papilloma had evidently ceased to grow the event might have been a different one.

Van den Wildenberg reports two cases, 17 and 18 months old respectively, operated on through the Killian tube spatula. These cases are interesting on account of the youth of the patients, but are too recent to be of value otherwise.

Zuppinger advises intubation instead of tracheotomy when interference for dyspnea is called for. His preference does not seem well taken because of the many cases in which the papilloma grows so high in the larynx that it would obstruct an intubation tube, but no less because the presence of the tube would act as an irritant to stimulate the papillomatous growth.

Ferreri favors laryngo-fissure for papilloma even in adults. He reports cases, two adults and one child. It seems very strange that anyone can still advocate this operation. In T. J. Harris's case, reported before this association in 1905 and 1906, in which the removal of a fibroma from the larynx of a child by laryngo-fissure was followed about a year later by the growth of a papilloma, the second laryngo-fissure for the removal of this growth was more justifiable than in the ordinary case of papilloma. But even in this case I should have been inclined to proceed as in any case of papilloma.

As to the cases which C. G. Coakley reported before this association a year ago, he said himself that they were too recent to tell whether there would be a recurrence or not. Consequently it must be admitted that his results as then reported cannot be considered as proof that one would be justified in operating without previous tracheotomy in every case. I think that he would admit that, at least in cases where dyspnea was marked, preliminary tracheotomy is the safe procedure.

What has been added to our knowledge of this subject during the past three years? As to the etiology and pathology, practically nothing. Wingrave's suggestion of the presence of adenoids as a causative factor seems to lack sufficient support to be worthy of consideration. Baumgarten's, that a gonorrheal discharge from the mother at birth may be an etiologic factor, is obviously, for many reasons a difficult question to investigate satisfactorily. That bad hygienic surroundings favor this affection is certainly borne out by its comparative rarity in the children of well-to-do parents. Wingrave states that papilloma is found more frequently in the anterior half of the larynx. In nine of my cases it is stated that the papilloma sprang from somewhere in the anterior of the larynx, but was limited to that region in only five of these. In five cases the site was one or both sides of the larynx and in three others the site is not definitely stated. In one case the papillomata grew from the interarytenoid space as well as from other parts of the larynx. On the whole my experience bears out this statement of Wingrave's.

Treatment.—In treating these cases our aim is to get rid of the papilloma in the shortest time and with the least risk to the patient. Secondary desiderata are that the voice should not be impaired and that there should be (in girls at least) no external scar. It is plain to any one who has studied this subject that no one method of treatment is applicable to every case. How, then, should one decide in a given case what method to pursue? The operation of laryngo-fissure I dismiss at once as never applicable to the treatment of this affection. The first question then which confronts one on seeing a case for the first time is shall we do a preliminary tracheotomy? Mackenzie advises tracheotomy as soon as complete aphonia is present. Garel operates without tracheotomy even when marked dyspnea exists. Others take positions between these extremes. I stated in my first paper that "these growths will not yield to any form of treatment which has been attempted, however radical, until their period of active growth has passed" or, in other words, until they have lost their power of reproduction. The age of the patient offers us little if any assistance in deciding whether the papilloma is still growing and has not lost its power of reproduction or whether it is in a quiescent state and will not recur if removed.

If, therefore, a case is seen before dyspnea has developed, a careful record should be made of the site and size of the growth and the patient should be kept under observation for a few weeks. If in that time there is no increase, removal without preliminary tracheotomy may be attempted. The various instruments used for operating with the aid of the laryngeal mirror must now give place to straight instruments and direct vision. This is obtained, under general anesthesia, the Jackson laryngeal speculum, the Killian tube-spatula or the Kierstein autoscope. I have had less experience with the first-mentioned instrument than with the other two, but find it offers distinct advantages where it can be used, not the least of these being the better illumination. For very small children, the Killian instrument is best on account of its size. After one of these instruments is introduced the larynx is sprayed with a 1 per cent solution of cocain to quiet the laryngeal reflex. Tracheotomy instruments should be ready for emergency. Great care should be exerted in removing the papilloma not to wound the mucous membrane, as any such trauma may be the site of a new papilloma. The advisability of operating without a previous tracheotomy in cases where there is dyspnea must be largely a matter of judgment in the individual case. If only a short time has elapsed since the first symptom and dyspena is already present, showing a rapid growth, a preliminary tracheotomy should be done. When the dyspnea is marked, or when without marked dyspnea, the growth is found to fill more than half the glottis, tracheotomy should also be done before any attempt at removal is made. After tracheotomy the papilloma should not be removed at once but the larynx should be examined from time to time to determine if the papilloma is growing or not. If, after a reasonable time, usually a number of weeks, there is no apparent increase the papilloma may be removed. If it recurs the operator should again wait until it has apparently ceased to grow before attempting a second removal. Repeated operations at short intervals are inadvisable not only because they are useless when the papilloma is still growing but also because they appear to stimulate the growth. While a cure is possible in a large number of cases by simple tracheotomy and time, as some of my cases show and as shown in a series of cases reported by G. Hunter Mackenzie, quoted in my first paper, removal of the papilloma when it has become quiescent hastens the cure and shortens the time of wearing the tube. Apart from the curative value of tracheotomy there can be no question that it makes the subsequent removal of the growth easier and safer. The risk of bronchopneumonia following, immediately or eventually, tracheotomy is a real one. But in the case of a large and rapidly growing papilloma or of a papilloma recurring rapidly after a removal without tracheotomy, this operation offers the only alternative to suffocation. Intubation I consider inadvisable in any case for reasons already stated. Proper hygiene undoubtedly lessens very much the dangers of bronchial complications. The home surroundings of the child should be very carefully considered. I feel confident that the outcome in two of my cases would have been different if the patients had had better home care. On the other hand I am inclined to believe that the presence of laryngeal papilloma alone makes the child more than ordinarily susceptible to bronchial troubles.

The total length of time which the tube has to be worn varies very much. Among my cases the shortest time was ten months, the longest time was four years and seven months. The shortest time between removing the tube and closing the tracheal fistula was two months and the longest time was one year. It is not necessary to leave the tube in long after the papilloma has disappeared, because after the tube has been worn a few months a fistula is formed which will remain open until closed by operation, so that a tube can be readily reinserted should it become necessary on account of recurrence. No fixed rule can be laid down as to the length of time which should elapse between the disappearance of the papilloma and the closing of the tracheal fistula. I am inclined to make the interval at least six months. The voice is normal in all my cured cases.

REFERENCES.

A. Neubauer: Archiv. f. Kinderheilk., Stuttg., 1905, xli, 21-28.

W. Koelreutter. Monatschr. f. Ohrenheilk., Berlin, 1905, xxxix, 501-506.

Garel. Ann. de Mal. de l'Oreille du Lar., etc., Paris, 1906, xxxii, 1-11.

F. N. McCreary. Laryngscope, St. L., 1906, xvi, 488-491.

J. Rogers. Laryngoscope, St. L., 1906, xvi, 488-491.

L. Turner. Jour. of Laryngol., London, 1906, xxi, 177.

W. Wingrave. Ibid., 215-221.

E. Baumgarten. Zeitschr. f. klin. Med., Berl., 1907, lxii, 272-283.

B. Robinson. Jour. of Laryng., London, 1907, xxii, 171.

Van den Wildenberg. Presse Oto-laryngol., Belge, Brux., 1907, vi, 346-351.

C. Zuppinger. Wien. Med. Wochenschr., 1907, lvii, 1030-1034.

Ferreri. Arch. Ital. di Otol., etc., 1905-6, xvii, 441-447.

T. J. Harris. Transactions of the Amer. Laryngolog. Ass'n, N. Y., 1906.

C. G. Coakley. Transactions of the Amer. Laryngolog. Ass'n, N. Y., 1907.

G. H. Mackenzie. Jour. of Laryngol., London, Sept., 1901.

XXXIII.

THE DIAGNOSIS OF THE OTITIC BRAIN ABSCESS.*

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Translated with special permission of the author,

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The relatively satisfactory and encouraging therapeutic results which have already been achieved in such a grave condition as brain abscess, have prompted surgeons, but more especially aurists, of every civilized land to be ever on the outlook for any new or improved methods which could contribute to an early diagnosis or a better operative technic.

Notwithstanding all previous endeavors, it often happens that the abscess is first discovered at the autopsy. Every practical surgeon can testify to the chagrin which one feels and the unjust reproaches which one heaps on himself under such circumstances, and on the other hand the real satisfaction one feels if he has been successful in recognizing and operating on a brain abscess in time. He thereby saves the life of the patient, who was unquestionably condemned to death, and his mental and spiritual welfare are advanced as well.

It cannot be denied that a marked advancement in this field of research can be shown, and that, through a proper appreciation of the symptoms and a careful analysis of the same, better results than formerly are now obtained.

The causes of a false or delayed diagnosis of a brain abscess of otitic origin are various. Every intracranial complication, which has its origin in a purulent otitis, may make its appearance in a sharply defined and easily diagnosed picture. For certainty of diagnosis however, a fully developed symptom complex is necessary. Let the contours of the picture be somewhat indefinite or in places effaced, then the diagnosis amounts to probability only.

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*Schwartze's Jubilee Number, Arch. für Ohrenheilkunde, Band 73.

Many symptoms which one attributes to a brain abscess belong perhaps to a complicating meningitis. When we reflect how intimately in the course of their development, the complications are intertwined, and how the symptoms of ear diseases, functional nerve disturbances and other brain diseases can closely simulate this condition, we are prepared to extract a diagnosis from inexplicable inconsistencies.

So great a master as Schwartze was uncertain of a diagnosis which seemed to lie between an abscess of the temporal region and a meningitis. The autopsy proved it to be a cerebellar abscess. Kuhn mentions a case where all the symptoms and especially the history of aphonia, pointed to a temporal lobe abscess of the left side. Instead, the operation and subsequent autopsy laid bare a diffuse meningitis. Panse observed a somewhat similar case. Not infrequently the symptoms speak for an abscess in the cerebrum, the autopsy proving it to be in the cerebellum.

The great variations and the commonly latent course of a brain abscess make the diagnosis often very difficult or even impossible. The greatest difficulty is experienced in those cases in which the latent period is complicated, the initial symptoms very slight, and the end stage comes on very quickly and follows a rapid course.

One often meets with patients, who because of an exacerbation of an acute or chronic ear inflammation, have certain symptoms which seemingly have no connection with brain involvement. The original disease disappears entirely, or is so much better that the patient thinks himself well and returns to his ordinary occupation. He is affected now and then with seemingly unimportant symptoms, perhaps a slight headache, upon which he lays no great weight and seeks no professional advice. If, however, he does consult a physician, the symptoms are generally made light of, or not correctly interpreted.

As an example of such a course, two cases are reported. A soldier who had passed through an attack of middle ear disease was discharged from the hospital as cured. Two weeks later he suddenly died. The autopsy revealed an abscess of the temporal lobe the size of a hen's egg. In the second case, a soldier who after 33 days' treatment was about to be discharged cured, suddenly developed symptoms of acute meningitis and died in a few hours. The autopsy showed a large left sided cerebellar abscess which had emptied into the cranial cavity.

In 10 per cent of cerebellar abscesses, Okada found that the patient died before any indication of intracranial complication had been found. In 14 per cent the symptoms were hidden by complications of an otitic origin, so that often only the latter were diagnosticated. In about 42 per cent the diagnosis was impossible on account of other intracranial complications, such as sinus phlebitis, leptomeningitis, and cerebral abscess which directly or indirectly were associated with the abscess in the cerebellum. Small abscesses in the cerebral hemispheres can cause marked symptoms only when they are in immediate proximity to the motor area, or near enough to have an indirect influence on it or upon some of the basal ganglia.

Not only may small abscesses remain latent, but also, as is well known, an abscess of the frontal lobe may reach an enormous size and be quiescent for a long time before any symptoms appear which give rise to a suspicion of its presence. Even in the temporal lobe, the presence of an abscess makes itself known only when its volume has so enlarged that a mechanical disturbance in the skull cavity takes place. The expanding mass causes a retarding movement on the blood stream within the cranial cavity, which is, as a general rule, characterized by pressure symptoms.

In three-quarters of the cases, fortunately, we can make an early and correct diagnosis, if we associate the brain symptoms, which are in themselves not characteristic but appear in other brain affections as well, with some pyogenic process which has preceded. Thus we must not overlook an existing or a preceding ear discharge which may be the cause of the brain abscess.

In every case therefore, where brain symptoms appear or a suspicion of the same is at hand, the present or past diseased condition of the organs of hearing must be thoroughly investigated.

Facts which should be considered as important circumstantial evidence may be stated as follows:

1. Diseases of the central cranial fossa usually lead to an abscess in the temporal lobe, seldom to abscess in other parts of the brain.

2. Diseases in the territory of the posterior cranial fossa lead usually to an abscess in the cerebellum.

3. Cranial bone disease may often be the causal factor of a brain abscess; diseases of the tympanic mucous membrane, rarely.

4. Men are more commonly affected than women in the proportion of 3:1 (385 males; 140 females).

5. The second and third decennium of life are most favorable for abscess. Their occurrence is rare before the fifteenth and after the sixtieth years.

6. In women past forty a brain abscess has seldom been found.

A very important consideration which contributes to the accuracy of the diagnosis, is the length of time that the patient has been under observation by the physician. The amount of the surgeon's experience is here, as in other realms of medicine, not to be under-valued.

In order to make certain the diagnosis of a brain abscess of otitic origin, it is necessary to recognize both its position in the cranial cavity, and the presence or absence of complications. Unfortunately it is almost impossible in all cases to give a typical picture for the course and symptoms of the disease. Some cases from the beginning take a stormy course with manifold symptoms of irritation and pressure, and lead quickly to death. Others take a very long and tedious course, and exhibit almost none or very few symptoms of value. Still others have a regular course and pass through all the phases. Cases in the last category are certainly very much less often observed than the atypical ones, and this remark applies especially to abscesses in the cerebellum. On this very account, the atypical cases play a very important role and their diagnosis now and then presents insurmountable difficulties.

In the course of a brain abscess two things are characteristic:

1. The symptoms appear scarcely ever alone.

2. They often present the character of an attack, the length of which varies greatly.

The remission of symptoms often covers such a long period of time, that it may be thought that the patient has recovered. In exceptional cases this intermission may cover a period of thirty years. Although in many cases they can remain about the same, the rule is, that each attack exceeds in severity that which has preceded it. The intensity as well as the number of the attacks may vary. Often the first will be fatal and then again it will be the third or fourth. Between the attacks the patient is only exceptionally free from symptoms, they gradually increase in number and severity until death takes place.

The attack, like the character of the symptoms, seems to depend on a sudden increase in the intracranial pressure, due to the expansion of the abscess and to the easily compensated circulation in its neighborhood.

The symptoms of an otitic brain abscess are, as is well known, usually classified as general and local. Von Bergmann divides them into three groups according to their cause and development.

(a) Those depending on or belonging to a pus forming process.

(b) Those depending on an increase in pressure or a disturbance of functions through displacement.

(c) Symptoms of central origin which indicate the position of the abscess.

In order to make clearer the picture of the development of a brain abscess we divide the course of the symptoms into three stages:

The *first stage* or period of the initial symptoms.

The *second stage* or the latent or manifest period, which is the period of the completed abscess and the symptoms depending on it.

The *third stage* or terminal period.

These three stages go hand in hand with the pathologic processes. The initial symptoms are the result of inflammation and vary as to their intensity. In many cases they are so poorly marked that they may be easily overlooked while in others they are relatively severe. The character of the symptoms is determined by the nature and position of the pathologic processes. Local manifestations depending upon the seat of the affection are more commonly wanting than is the case with brain tumors. This fact has a two-fold explanation: In the first place the abscess is situated in a part of the brain, the temporal or frontal lobe, where local processes, of whatever nature they may be, cause but little local effect. In the second place the slow development of an abscess causes less disturbance than the pressure of a tumor.

A positive diagnosis of a brain abscess in the inflammatory period or in other words during the initial stage or period of development is much to be desired but almost always impossible. This is explained by the fact that the symptoms during this stage are generally overshadowed by the symptoms of the accompanying ear disease. A failure to make a diagnosis at this time is without danger to the patient. The full development of the abscess is not completed, the operation would probably be without results, and would deter the surgeon from again submitting the patient to an additional procedure. It is hard to conceive of either a surgeon or an aurist who would seek for a brain abscess in the course of a stormily running middle ear inflammation.

When one considers that Schwartze found only eight brain abscesses in 8,425 cases of middle and internal ear disease, and Jansen only seven in 5,000 cases of inflammatory middle ear diseases, and that this is the experience of all who have interested themselves in the matter, we can see why there should not be a suspicion of a brain abscess during the course of an acute or chronic middle ear attack. If following a poorly marked initial stage, a shorter or longer latent period without symptom follows, it should not be a matter of surprise if the disease remains unrecognized or is only barely suspected.

In order to recognize any given disease one must, as a matter of course, have a certain symptom complex. The same holds true for a brain abscess. In order to recognize the condition, there must be certain symptoms, and in order to make a positive or even a probable diagnosis, general and local symptoms of a specific type must make their appearance. If, however, we wait for the fully developed picture as is given in most text books before beginning a surgical procedure, our therapeutic results may have a very sad ending.

Of course the presence of one or two symptoms should not result in a positive diagnosis, but to wait for all the general symptoms and still more for the local symptoms, is superfluous and dangerous for the patient. Looking back over the history of the case, and considering the principles already laid down, there are always a few symptoms of a more or less characteristic nature which stand out prominently.

By an analysis of the symptoms which accompany a brain abscess, we may conclude: (1) That, in contradistinction to the symptoms of brain tumors, where local manifestations are above all the rule, the local symptoms are not in themselves characteristic but the general manifestations occupy a more important place. (2) That in a large number of cases a certain symptom complex is constantly present, while in others it only partly appears. There are also cases where symptoms are observed, which do not generally appear with brain abscess but only here and there occur in the very rare cases. We are not certain if they are really manifestations of the brain abscess or belong to some other complication in the cranial cavity. Local symptoms, which are dependent on the involvement of a certain part of the brain, the motor area, or the cranial nerves, may appear during the course of the disease, may be absent altogether, or may be deferred until just before death. Usually they belong to the later evidences of the abcess, and when present make the diagnosis and localization of the trouble easy. According to the present standpoint of the science there is no uniform symptomatology of an otitic brain abscess.

In the initial stage, in which the inflammation speaks for the dvelopment of the abscess, one observes that a patient, who has suffered from an acute or a chronic middle ear inflammation, or who as a sequel to these troubles has had a mastoid operation performed, will suddenly develop pain in the diseased ear or in the corresponding side of the head. Not infrequently no ear pain is present, or it is minimal; the patient complaining only of the pain in the head. Perhaps he has had one or more attacks of vomiting without accompanying nausea. In the beginning of the ear exacerbation, chills or perhaps only a chilly sensation will be observed. The chills vary not only as to their intensity but also as to their length. Repeated chills force one to the conclusion that a septic process, most likely a sinus phlebitis, has developed from the abscess. If the chills develop regularly with a fixed pause between, we have to do with a malarial fever or some complicating disease (Macewen). Generally speaking, repeated chills with a brain abscess are rare. In a child of five years in whom the author had opened an abscess in the temporal lobe, a remittent fever developed. There was naturally some fear that pyemia had developed, but further observations showed that the abscess was complicated with malaria.

In the initial period of an uncomplicated abscess the temperature is only slightly elevated (37.7 to 38.3° C.) and with it we usually find a delayed pulse, coated tongue, and general weakness. All of these symptoms, not excluding dizziness, may accompany a middle ear inflammation without an intracranial complication coming into consideration.

The initial period varies from a few hours to as many days. If the abscess passes without symptoms into the latent period, the patient is usually lost to observation until later, when marked symptoms develop. Not infrequently the initial period passes directly over into the next, where symptoms of intracranial pressure and disturbance of the cranial cavity are made manifest. The majority of brain abscesses come under observation during this stage.

It is a self-evident fact, that to make a certain diagnosis of brain abscess, an exact knowledge is absolutely necessary of all of its symptoms as well as the symptoms of those diseases which complicate it, or can be mistaken for it. A brief recapitulation of the symptomatology of the otitic brain abscess will therefore not be out of place.

The patient during the second or manifest stage, gives one the impression of a very sick person. This impression is a peculiar one. The color of the skin is pale yellow and earth like. The patient is apathetic, sleepy, gazes dreamily into emptiness and an inability for mental concentration is a regular accompaniment of the sluggishness of the cerebral functions. He answers ordinary questions unwillingly, and if a long question is framed he will often sleep before it is completed. He may answer the first part of the question correctly, then the words follow one another more slowly and articulation becomes indistinct. Speaking tires him greatly. The sleepiness very greatly resembles opium poisoning (Macewen). As the disease progresses the stupor increases until it finally becomes difficult to arouse the patient even for a moment.

The ability to use his strength gradually diminishes until it is finally lost. The patient prefers to lie in the horizontal position only. The taking of the side position is very seldom observed. Changes of position almost always bring on attacks of dizziness, nausea or even vomiting.

Many patients are loquacious. In rare cases one sees a mental unbalance of such a degree that the case will be admitted to an asylum. The intelligence is usually less affected than in other brain conditions; notably meningitis and softening of the brain (Lebert). Loss of consciousness in the course of a brain abscess is not usually seen until just before death. In other cases it is intermittently somewhat clouded and, in the few days just preceding death, again becomes perfectly clear. There are cases with attacks of loss of consciousness but in which after a few hours the mind becomes perfectly clear. In abscesses of the cerebellum, the consciousness is hardly ever clouded and scarcely ever lost, and is extinguished only at the end of the disease. The clouding of the consciousness may increase to stupor or even coma. Delirium and melancholia with a tendency to suicide may be present.

The first and almost never-failing symptom of intracranial pressure is headache. In rare cases it may be absent. Generally it is the first and most important and constant symptom which accompanies a developing or developed abscess. The headache may be constant, remittent, or intermittent in character. As a rule it indicates the seat of the abscess but this is subject to many exceptions. Cases are known where both surgeons and aurists depending on this symptom have erred in the location of the abscess, not fiding it during the operation but at the autopsy. It thus happens that the abscess may be found in the cerebellum, while the headache is localized in the temporal region. The contrary may be also true. Exceptionally frontal headache is seen with abscess of the cerebellum. Körner cites five cases of abscess in the temporal lobe, in which the pain was usually referred to the back of the head.

The headache is of varying intensity from a slight heaviness to a pain which is almost unbearable. It may be limited to a circumscribed region or involve the whole head. Everything which increases pressure within the cranial cavity, such as alcoholic drinks, overheating, straining at stool, mental work, etc., brings forth or intensifies the headache. Children usually complain of a diffuse headache. Percussion of the head usually elicits pain at the seat of the abscess. Macewen has noted that the nature of the contents of the cranial cavity has an influence on the character of the percussion note, and Knapp heard a stronger tone on the diseased than on the well side. In the case of a cerebellar abscess the headache is generally located in the back of the head. This is an important symptom in differential diagnosis and will be observed in over half of the cases. Often, however, the pain is not fixed. Okada noted pain of a fixed type in 54 per cent of cerebellar abscesses. Politzer regards headache in the back of the head as the one important symptom of a latent cerebellar abscess

The headache exercises, very often, a characteristic influence on the movements and position of holding the head. (R. Müller.) The movements of the patient are careful and strained, in part as the result of cerebellar ataxia, in part to avoid shaking the head. Naturally this symptom is only of value so long as the patient is able to be about. The head will mostly be held stiffly and without movement. When the pain is in the back of the head, the position is backward and toward the affected side. The neck and back will also be held somewhat toward this side and backward. Providing a chronic inflammation of the ear is known to be present, such a position of the patient will enable the physician to make a diagnosis of a cerebellar abscess, even if he only sees the patient across the room. Hyperesthesia of the head has been seen only three times.

A rigidity of the sternocleidomastoid muscle, with pressure pain in the deep tissues and along the course of the jugularis, will be found in abscess of the temporal lobe or of the cerebellum, complicated with a thrombus of the sinus transversus.

The lowering of the pulse rate, during this period, is a symptom of great weight and immeasurable importance. It may fall as low as 30 to the minute, but usually the frequency is from 48 to 60. The pulse does not correspond to the temperature. A slowing will be observed in other brain affections, notably tumors, and in encephalitis, as a result of the elevation of intracranial pressure. There comes a period during the course of a brain abscess which is of great diagnostic importance. If the temperature is high and the pulse is slow, one can with certainty make the diagnosis of an intracranial trouble and at the same time exclude a bodily sickness as the cause. If at this time an ear discharge is present which has brought on the intracranial disease, then the temperature indicates an encephalitis or meningitis complicating a brain abscess. A meningitis. caused by an infection or an infectious sinus thrombosis, gives usually a small and quick pulse. In such a case the character of the pulse depends not on the brain abscess but on the infection.

The lowering of the pulse rate is not always in proportion to the size of the abscess; small abscesses, as well as the larger ones, have not infrequently a distinct slowing action on the pulse. Perhaps the increased intracranial pressure is the principal cause of the slowing of the pulse rate, for after the opening of a large abscess, the rate will be surprisingly increased.

In many cases of brain abscess only a slight increase in the intracranial pressure is noted, which is explained by the fact that the collection of pus takes but little more room than the destroyed brain substance, and naturally there will be only a slight slowing of the rate. Von Bergmann found that the lowering of the rate is more frequent in cerebellar than in cerebral abscess, but the author can find no difference in this regard. The difficulty of diagnosis is made greater if there happens to be a preexisting brain affection in the neighborhood (Schwartze). On the other hand, the simple lowering of the pulse rate may lead one into the error of believing an abscess is present (Koch). The author had lately the opportunity of observing a very much run down child of 9 years, who had a chronic middle ear discharge and complained from time to time of a right sided headache. The pulse was 42 to the minute. The lowering of the pulse rate was entirely due to an accompanying heart affection. Irregularity of the pulse rate is sometimes observed in cerebellar abscess.

During the latent period symptoms of dizziness are almost never absent, and are usually more marked in cerebellar than in cerebral abscesses. The dizziness is of various kinds and various degrees of intensity; changes in the position of the head or increase in the headache cause it to appear when it is otherwise latent.

Vomiting, during this period, is an almost ever present symptom. This applies to other intracranial diseases as well. In the case of a brain abscess, the symptom appears as soon as the bed-ridden patient gets up or attempts to walk about.

In the case of cerebellar abscess, vomiting continues longer and is much more common. It belongs to the most important symptoms of this disease, and has been observed in over 75 per cent of the cases. Koch mentions one case where vomiting was mistaken for a sign of pregnancy. The autopsy revealed the error and showed that the patient was not pregnant. Large abscesses, with difficulty in swallowing, indicate a pressure on the pons.

A common symptom of brain abscess is a disagreeable odor from the mouth. The tongue is usually thickly coated, often dry, brown or cyanotic, and trembles when extended. The teeth are usually incrusted. The odor from the mouth resembles very closely the odor from the ear discharge.

We have, however, the same fetor in a neglected middle ear process without an intracranial complication being present. In the case of an infected sinus thrombus the odor is, if anything, more marked than is the case with a brain abscess.

As a rule we find marked constipation, which is also observed in tumors and meningitis. In sinus thrombosis, diarrhea is the rule. There is loss of appetite, but less often an excessive desire for food. The patients have often such a strong objection to taking nourishment that a resort to artificial alimentation is necessary.

A high degree of emaciation, as a general rule, is commonly observed in the latter part of this period. This condition, together with constipation, headache and slowing of the pulse makes up a symptom complex that is of great diagnostic value. Emaciation from high fever, chills, sweating or diarrhea, speak for a general infection and not a brain abscess.

The breathing is slowed, but usually regular. In cerebellar abscess the breathing is far more slowed than is the case with abscesses of the cerebrum. Exceptionally, Cheyne-Stoke's breathing or a decidedly irregular type is observed. If there is a pressure on the medulla, there may be a cessation of the respiratory function, long before the heart ceases to beat. In meningitis and infected sinus thrombosis, as a rule, the respiration is accelerated, and only when the posterior cranial cavity is encroached upon by the meningitis, does the respiration take on the type of a brain abscess.

Retention of urine will occasionally be observed. Commonly there is found a small quantity of albumin, which disappears when the abscess is opened. Polyuria and glycosuria has been observed in a small number of cases.

Chills during this period are very seldom noted. They usually occur when a new abscess forms on the periphery of the old one.

The temperature continues, throughout the course of the latent period, very nearly normal. For a brain abscess this is absolutely typical, and is in direct contradiction with leptomeningitis and all other brain complications having their origin in the ear. In many cases the writer has noted a temporary and inconsiderable increase. A marked and permanent increase speaks always for an intercurrent complication. Oppenheim makes the statement, that where one observes a marked and persistent increase in the temperature, the probability is that there is no abscess present, or at least the abscess is a complicated one. In the initial and final stages of the abscess one does, however, note a slight increase but of short duration only. Koch shares in this opinion as regards cerebellar abscess. Okada observed in half the cases of cerebellar abscesses a marked increase in the temperature throughout the entire course. This is only natural when one considers that a cerebellar abscess is very seldom uncomplicated.

Optic neuritis, which is always a striking symptom where increased intracranial pressure is present, takes an important place in the symptomology of brain abscess. It belongs to the more common of the manifestations of a large abscess toward the end of the second period, and the author has noted it even as early as the beginning of this period. As a rule the neuritis is not far reaching and is seldom so clearly marked as is the case with brain tumors. An atrophy of the nerve seldom follows the neuritis. The author noted this sequence in a syphilitic where every symptom pointed to an abscess, but neither by operation nor autopsy was the diagnosis confirmed. Usually the neuritis is more marked in one eye than the other. The side of the brain affected is not always indicated by its development, for crossed neuritis has been observed. When the development of the abscess is very rapid there is not time enough for the development of the neuritis; this is also the case in cerebellar abscesses, where the surrounding inflammation is not great. (Macewen.) The writer has noted an exception to this rule. In one case, the neuritis appeared eight days after the beginning of the symptoms which pointed to an abscess. The abscess in this case was acute and passed directly over into the manifest stage. Hinsberg notes neuritis optica much more commonly in cerebellar abscesses (66 2-3 per cent). In other intracranial processes, meningitis, encephalitis, tumors, etc., the same condition is observed. It has also been noted in an ordinary uncomplicated middle ear discharge. In these cases it follows, through the involvement of the plexus caroticus of the sympathetic, which in turn leads to a vasomotor disturbance of the optic nerve and retina. The last point has not been proved. Macewen believes the cause to be a low grade of meningitis depending on the ear discharge.

Amaurosis has been observed in a single case of cerebellar abscess.

Changes in the motility of the pupils are noted following the increased pressure. In the case of temporal or frontal lobe abscess a sluggishness of action may be recognized on the diseased side. A sluggish pupil, myosis, or mydriasis occurring on the same side as the ear discharge gives additional evidence that a collection of pus is to be sought for on the corresponding side. Sometimes one pupil is enlarged and then again it is the other. The pupil symptoms are very inconstant and a normal or contracted pupil does not exclude a cerebellar abscess. Generally speaking an enlarged pupil on one side or both sides indicates some space-occupying process in the interior of the cranial cavity. In diffuse meningitis, the pupils are at first equally contracted until the pressure symptoms appear, when mydriasis with sluggish pupils is the rule. An infected brain thrombus, with the exception of the thrombus of the sinus cavernosus, is seldom followed by disturbances of the pupils. Strabismus as well as a fixed position of the bulb is seldom seen. Ptosis more often occurs with a temporal lobe abscess than it does with a cerebellar abscess. Exopthalmus is rare and usually occurs with cerebellar abscess complicated with a sinus thrombosis.

Convulsions during the latent period are uncommon and are not characteristic, and when seen are usually found in children. The convulsions depend upon a secondary irritation of the motor tract. Severe chills are often mistaken by the laity for convulsions (Macewen).

Local symptoms may entirely fail, be poorly marked or make their appearance late. They depend on the size and location of the abscess, and have a varying value in the diagnosis. They mean much if the abscess is in the region of the motor area and very little if it is located in the frontal occipital or temporal lobe. They depend upon the destruction of the brain substance or its softening, or on the edema which precedes its enlargement. This edema may recede, and with it a disappearance of one or more of the local symptoms. The pus, in the peripheral positions, presses the nerve paths apart without causing a solution of their continuity. If the gray substance remains intact, the abscess may reach a wide limit, stretching over almost the whole hemisphere without the appearance of a single local symptom. Just in proportion, however, as it approaches the cortex or inner capsule, the local symptoms make their appearance. As an abscess can occur in any part of the brain, the local symptoms must agree with those general conditions which are characteristic for the different brain regions.

The appearance of deafness of a central origin, which can be total, in the opposite but previously healthy ear, is a very important local symptom. Usually it is impossible in the apathetic or somnolent patient to test the hearing of the sound ear, for it goes without saying that in order to determine this point we must be in position to test the condition of the integrity of the peripheral organs of hearing.

Disturbances of speech, for example, agraphia, anarythmia, optical aphasia, picture aphasia, and motor aphasia are common local symptoms. These different forms of speech disturbances can only take place when certain limited parts of the left hemisphere are involved. In cerebellar abscesses, the disturbances of speech are not exclusively due to involvement of the fibres of the nucleus in the bridge, the medulla or temporal lobe, but to a general disturbance of the intellect (Körner), or mechanical disturbance such as rigidity of the masseters. (Macewen.) A temporary loss of speech may take place. A certain psychical disturbance may be present, making the discovery of the aphasia difficult.

Cerebellar ataxia or dizziness depends upon a destruction or irritation of the vermis, from an abscess of the hemispheres which has influenced the same or from a collection of pus over the tentorium (Nothnagel). This symptom is only of value if the sensorium remains free and if labyrinth involvement can be excluded. It is a symptom which often accompanies a cerebellar abscess, as Jansen has noted in more than 80 cases. Poulsen and Heimann have shown that this symptom may be absent even when the vermis is diseased. On the other hand, we can have ataxia in hysterical patients with accompanying ear disease (Oppenheim). If we consider that in many cases, on account of the severity of the sickness, the ataxia can not be recognized, and that disturbances of equilibrium as a result of dizziness are difficult to differentiate from ataxia, this symptom when considered in the light of the demonstrated cases can not be given great weight. It is certainly not pathognomonic for abscesses of the cerebellum as the same thing has been noted, in five cases of cerebral abscesses, in meningitis serosa and meningitis purulenta.

In addition to the positive group of symptoms, which are due to certain localized disturbances in the brain, a brain abscess gives rise to a second group, the so-called distal effects. These remote or distal effects are the result of the influence of the brain irritation on another area in the brain, in a manner about to be stated. These remote effects, like the original lesion, give rise to certain definite and characteristic symptoms of the same character as has already been described. As is well known, they may arise as a result of an inflammatory edema which surrounds the abscess in a more or less broad zone or as a result of the increased tension of the cerebrospinal fluid which is in turn due to the increased pressure in the cranial cavity. This tension may be equalized either through the fluids in the ordinary way or through the semi-soft mass of the brain itself. Those parts which are in the immediate vicinity of the temporal lobe will be more affected than the parts farther removed.

The distal effects of a temporal lobe abscess do not extend beyond the tentorium. The effects are also limited in other directions except toward the capsula interna. If the capsule is involved, crossed pareses of the extremities, more seldom crossed paralyses and convulsions, and tonic cramps of the crossed side occur.

The hemiplegia in many cases is manifestly the result of the heightened pressure, and there is a return of the normal functions as soon as the pus cavity is drained. In other cases the manifestations are explained by inflammatory processes.

It is very important to make certain in just what order the various parts of the affected side were involved, so that one may make out whether the motor center in the cortex or the conducting path in the capsule is part of the pathologic process. If the former, the face, then the arm, and lastly the leg is affected, without a disturbance of the sensory functions, the probability is that a temporal lobe abscess has exercised its distal effects on the gyrus centralis, broadening out from below upward. The pareses are usually on the crossed upper extremity and more marked than when the legs are affected. Occasionally, a crossed hemianesthesia and a homonymus bilateral hemiopia will be observed.

Anosmia of the same side is of very rare occurrence. With it a paresis in the territory of the crossed facialis may occur, but a spasm of this nerve is seldom seen. The paresis is the result of the distal effects of a temporal lobe abscess on the inner capsule, a cerebellar abscess affecting the bridge, or the distal effects of an abscess of the bridge itself. If the paralysis of the facialis occurs as a result of a destructive process in the middle ear or mastoid process, or follows from the pressure of a cerebellar abscess upon the entrance point in the porus acusticus internus, then the effect is noted on the same side.

It is necessary to differentiate between a paralysis of the facial muscles depending on a lesion of the cortex and an inactivity of the facial muscles depending on a paralysis of the facial nerve. In a cortical lesion an advanced paralysis is seldom observed. The patient can close the eye and retains to a certain extent the ability to show expression. The sensation of taste remains in the anterior end of the tongue. (Macewen.)

The distal effects of the cerebellar abscess usually involve the medulla oblongata and cause death through paralysis of respiration. If the abscess is in the cura cerebelli or the bridge, it can in addition to respiratory paralysis cause a paralysis of the crossed extremities, crossed facial paralysis, paralysis of both legs, homonymous hemiplegia, trismus, and weakness or paresis of the arm or both legs on the same side. One will also observe in an abscess of the bridge, copora striatum or optic thalamus, anesthesia and loss of muscular power in the extremities of the other side, perhaps due to an involvement of the posterior portion of the inner capsule A small abscess in this region may run its course without symptoms until a rupture takes place (Gowers). An alternating hemiplegia has been noted in abscess of the bridge.

Stiffness of the neck, with retraction of the head and a slight degree of opisthotonus, has been noted in an uncomplicated cerebral and cerebellar abscess (Moss, Hinsberg). More often such symptoms will be observed in meningitis, extradural abscess of the posterior cranial fossa, and in a purulent inflammation of the pons or a ruptured abscess affecting the medulla.

The deep and superficial reflexes are so irregularly affected, that no positive statements as to their value can be made. The patellar reflex varies; at times it is increased, at times diminished. and at times normal on both sides. In many cases it is absent on the affected side. Koch consideres the failure of the patellar reflex on the affected side, as an important differential point in favor of a cerebellar abscess as against a temporal lobe abscess.

Fibrillary twitchings of the muscles, although not at all pathognomonic for a brain abscess, may appear toward the last end of the latent period and in the final stages may be well marked.

A partial, but seldom total, paralysis of the oculomotorius of the affected side may be brought about when the temporal lobe abscess is of sufficient size. Usually mydriasis and ptosis with, at times, a paralysis of the rectus superior and internus, is noted. When a total paralysis of the oculomotor nerve is present we have strabismus, fixed pupil, and paralysis of all of the external eye muscles with the exception of the superior oblique and the rectus externus. The movements of the ball are, therefore, impossible with the exception of a slight excursion away from the median line and downward. As has already been mentioned, the oculomotor can be involved in a sympathetic inflammation with a meningitis.

Macewen states that when the following symptom complex is noted we can say with great probability that the abscess is located in the temporal lobe: 1. When the oculomotor paralysis is on the same side as the lesion; 2. When a crossed hemiplegia is present which starts in the face and whose characteristics indicate a cortical origin; that is when the paralysis is noted most plainly in the face, the arm being but slightly and the leg scarcely or not at all involved; 3. When a disturbance or loss of the sensations is at the same time absent.

Abducens paralysis of the same side will be noted in cerebral and cerebellar abscesses; in the latter case also a crossed paralysis of this nerve.

Trigeminal neuralgia and paralysis of the hypoglossus are rare conditions with temporal lobe abscess. Generally speaking, such affections of the cranial nerves, develop as the result of bone disease or meningitis rather than from a brain abscess.

The following symptoms have been mentioned in the literature, but their occurrence is seldom: Sensitiveness to light, conjugate deviation, nystagmus, singultus, retention or incontinence of urine, crossed pain and neuralgia of the ischias.

The organ of hearing exhibits during the course of an abscess, symptoms of an acute or chronic ear discharge, cholesteatoma, granulations, symptoms of pus retention, inflammatory softening of the mastoid bone, etc.

There has been noted, in very exceptional cases, a spontaneous empyting of the abscess, by means of a fistulous opening to the external canal or directly through the cranial bones. One almost always sees at least a trace of the preceding ear disease, but almost never a perfectly normal drum. In the vast majority of the cases, the cerebellar abscesses are found in combination with a purulent inflammation of the labyrinth. Corresponding to the changes in the labyrinth, the cerebellar disturbances of function are of a labyrinthine nature. The tests for hearing can not always be relied upon to give us an exact differential diagnosis, for functional disturbances are not infrequently found with a cerebral abscess, and then again they may be entirely wanting in the cerebellar type. In doubtful cases, however, these tests may give us a very important starting point for our diagnosis.

When but the one ear is involved, there may appear, in rare cases, a sudden deafness in the opposite or sound ear, or a sudden improvement on the affected side. (Schwartze, Lucae, Herpin.) Körner was the first to explain the phenomena by the fact that the hearing center for the opposite ear, was situated in the temporal lobe. With a one-sided ear discharge, the sudden appearance in the previously sound ear of deafness of central origin, is a decisive symptom in the diagnosis. As this phenomenon has also been observed in the case of cerebellar abscess, Schwartze's observation, that it is the result of marked hydrocephalus internus or a hyperemia of the labyrinth, is the more nearly correct explanation.

It has been sought to prove that a shortened bone conduction has some value in the diagnosis of a cerebellar abscess, but Körner has convinced himself of the untrustworthiness of this symptom, as he has often observed it in the case of temporal lobe abscess and often found it wanting in the cerebellar type.

After the termination of the latent period, which occupies a varying length of time, the so-called terminal stage makes its appearance. This period, either through the rapid increase in the size of the abscess, through its deleterious influence on the vital parts of the brain, or through its emptying into the lateral ventricles or upon the brain surface, leads quickly to death with severe symptoms.

The patient usually dies of a severe and fatal meningitis, or the picture just before the end is that of an apoplectic stroke. Stupor and coma may come on gradually or death may be sudden. At times a leptomeningitis may develop without the discharge of pus, in which case the inflammatory zone has reached the pia. If the course up to this time has been entirely latent, or even if some of the symptoms already spoken of have been present, this has no influence on the picture of the end stage. The patient will suddenly complain of chills and fever, the headache will increase to a great intensity, there will be one or more vomiting attacks, and then collapse, loss of consciousness, small slow pulse, Cheyne-Stokes' breathing, or perhaps complete paralysis of respiration, enlargement and immobility of the pupils, coma, stupor, and death in a few hours. In isolated cases an intercurrent disease may be the cause of death. Abscess of the temporal lobe more commonly than all others, breaks through into the lateral ventricles, and therefore the terminal stage follows the latent without an interval.. This is the reason why a certain portion of these cases are only diagnosticated at the autopsy, or it is only just before death that the symptoms are recognized.

The terminal stage lasts usually about twenty-four hours, seldom longer.

Lumbar puncture as an aid in the diagnosis of the otitic brain abscess is next to be considered. This procedure is dangerous, and because of the sudden change it brings about in the intracranial pressure and the change in the amount of fluid in the cranial cavity, it may induce a sudden rupture of the abscess, and be the cause of an instantaneous death. This has often been seen in the case of brain tumors, where death occurred suddenly through respiratory failure, or paralysis. The lumbar puncture may be the cause of the breaking of the newly formed adhesions with the pia, and allow the enclosed pus to escape on the surface.

If therefore we keep these facts in mind, and also the fact that by the history of the case, we can usually arrive at a definite conclusion, this procedure must be limited to a very few cases, principally in those where the symptoms are not well marked and where it is important to differentiate between an abscess and a meningitis purulenta. Here the procedure is of undoubted value.

So far as our knowledge goes the results of the lumbar puncture have given us the following data:

1. Cloudy fluid, with pus cells or bacteria, awakens the suspicion of brain abscess.

2. A very heavily clouded fluid with many pus cells and bacteria suggests a purulent meningitis.

3. A very slightly clouded fluid with bacteria, points to a disease of the pia, but one is not certain whether we have to deal with a diffuse or circumscribed process of the meninges. A fluid of this character was observed in a cerebral abscess

and led to a very sad result, as the abscess remained unopened. (Brieger, Ruprecht, Wolf.)

4. If the fluid is free from pus cells and bacteria, then no purulent meningitis is present or we have to do with a very circumscribed process.

5. Meningitis serosa or an encapsulated purulent meningitis is to be thought of when the fluid is increased in quantity or stands under pressure.

6. An opalescent fluid will be probably due to a meningitis tuberculosa even if the tubercle bacilli cannot be found.

Although we seem to have so many certain starting points for our diagnosis of temporal lobe abscess, we are not always protected from making a mistake. The most characteristic symptoms may lead us into error. Circumscribed headache, pain on percussion in the temporal region, or the posterior part of the head, neuritis optica, dizziness, vomiting, and the general condition of the patient indicated in many cases an abscess where the autopsy proved a purulent meningitis to be the real trouble. (Clinic at Halle.) In other cases the disturbances connected with the brain were ambiguous or the clinical picture of the abscess was clouded by intercurrent complications. Comparatively speaking, the temporal lobe abscess, by a consideration of its symptoms and history, is the most easily diagnosticated. The points of selection for the brain abscess are in the large majority of cases in the temporal lobe or cerebellum.

Abscesses of the frontal lobe, which are generally rare (3 frontal, 362 temporal), fail to give any definite symptoms. This is also the case with brain tumors in this region. The first symptoms show themselves through the distal effects after the abscess has reached a certain size: Disturbances of speech if it is left-sided, and monoplegia of the opposite facialis or the arm. Occipital lobe abscesses are also very rare, and according to von Bergmann, are usually the extension of an abscess having its origin in the temporal lobe.

In children, all intracranial processes, as well as an ordinary middle ear inflammation, usually develop with symptoms of irritation and take a more active course than is the case with adults. In children an ordinary extradural abscess may be mistaken for a brain abscess. (Körner.) Local symptoms are seldom or never well marked.

The diagnosis of the cerebellar abscess presents, generally speaking, far more difficulties than is the case with a temporal lobe abscess. The picture is poorly marked and not clear, and the diagnosis amounts to a probability only. Often the real facts of the case are only possible, when one can study the wound conditions during the opening of the mastoid or the exposure of the sinus.

Koch divides the clinical picture of the cerebellar abscess into three types:

Those cases in which marked and manifold local symptoms allow us to make a diagnosis.

Those cases with marked brain symptoms, but with few and poorly marked local symptoms.

Those cases in which neither the general nor the local symptoms are well marked, but the diagnosis is made possible by the course of the purulent inflammation, the finding at the mastoid operation and the presence in and through the dura of a fistulous opening leading to the discovery of an abscess.

It is possible to determine beforehand, whether two abscesses are present at the same time or whether one is situated in the cerebrum and the other in the cerebellum. If the symptoms do not entirely disappear after the operation and if no purulent symptoms are present to explain the remaining symptoms, we must be suspicious of a second abscess, whose location must be determined by the symptoms formerly present.

Where symptoms of intracranial complication are present and the differential diagnosis lies between a temporal lobe and a cerebellar abscess, the following points are to be taken into consideration:

1. All general symptoms are common to both locations with the following exceptions:

(a) With temporal lobe abscess it usually locates the seat of the trouble. With cerebellar abscess, it is usually located in the back of the head and sometimes in the forehead.

(b) Disturbances of the eye muscles.

2. In the cerebellar abscess, as the greater proportion of the deaths are due to failure in locating the abscess early enough, we must not wait for the special and local symptoms of the abscess in the hemispheres.

3. Naturally a cerebellar abscess may also exhibit characteristic distal effects, which consist of a disturbance of the function of the nucleus and the stems of the last six cranial

nerves, but these phenomena are far less infrequent than is the case with tumors.

4. Disturbances of equilibrium, gait, breathing, motor disturbances of speech, stiffness of the neck, trismus, nystagmus, paralysis of the facialis of the same side, paralysis of the extremities, amaurosis without atrophy of the optic nerve, convulsions in children, are all symptoms of the cerebellar abscess.

5. In cerebellar abscesses the disturbances of speech have the bulbar character.

6. Crossed, pareses, paralysis, spasms, often convulsions, hemianesthesia, amnesic aphasia, paraphasia, optico-acustic aphasia, aphasia for words, agraphia, hemiopia, ptosis, abducens paralysis, total paralysis of the oculomotorius, usually indicate a temporal lobe abscess.

7. In two-thirds of the cases of cerebellar abscesses the local symptoms are absent or poorly marked.

8. The temporal lobe abscess as a rule causes more or less local phenomena.

9. The temporal lobe abscess is most often found connected with caries of the tegmen tympani; the cerebellar abscess with caries of the posterior wall of the petrous bone or with a purulent inflammation of the labyrinth.

Here and there cases may be noted in literature where, after a collection of pus in the brain has been diagnosed, the one type of abscess was mistaken for the other. (Drumond, Garngee, Barr, etc.) Hansberg, in the case of a temporal lobe abscess, which was located in the inferior and posterior part of the lobe, maintained the diagnosis of cerebellar abscess. The autopsy showed that it was indeed a temporal lobe abscess, but lay quite close to the cerebellum.

The difficulty is increased if we have to deal with a double-sided ear discharge, which in the case of a cerebellar abscess can present insurmountable difficulties. If disturbances of speech are present, then, ceteris paribus, there is an abscess of the left hemisphere. The location of the pain, the percussion tenderness and the paralysis on the opposite or the same side, are of great diagnostic value.

The differential diagnosis between abscesses of the cerebellum and a purulent inflammation of the labyrinth is immensely difficult. Both diseases may develop together and have similar symptoms. To these belong dizziness, nausea, headache, vomiting, nystagmus, changes in the temperature curve, etc.

Neuritis optica is common in cerebellar abscess, but exceptional in labyrinth disease. It may, however, be wanting in the former trouble. Hemiataxia and hemi-anesthesia, are never seen in purulent labyrinth disease. Disturbances of equilibrium may be wanting in both diseases.

H. Neumann considers nystagmus a very important symptom in the differential diagnosis. In abscess of the cerebrum it is almost never present. In cerebellar abscess, horizontal nystagmus has been noted both toward the diseased and toward the well side. It increases in intensity as the disease progresses to a far greater degree than is ever noted in labyrinth disease. Neumann and Barany made the important observation, that in the beginning of the labyrinth disease the nystagmus is toward the diseased side, but that later it entirely disappears. In the beginning of a cerebellar abscess, the nystagmus is toward the well side, then it may suddenly change toward the diseased side. If one observes this latter symptom, the diagnosis of a cerebellar abscess is positive, and the labyrinthine origin of the nystagmus is excluded. Besides if a cerebellar abscess is combined with a purulent labyrinth disease, the intensity of the nystagmus quickly diminishes after the labyrinth operation, if it is of labyrinthine origin, and is not at all affected, if it is due to a cerebellar abscess. The cerebellar nystagmus has a rhythmical character and has the identical features of the labyrinth type.

The differential diagnosis of the extradural abscess is in most cases not difficult. General symptoms met with are: malaise, general outward impression of a very sick person, weariness, dizziness, vomiting, etc., which are also seen with abscess. Usually these symptoms are all absent. Changes in the pulse rate are very rare. If the rate is lowered, the size of the extradural abscess is great enough to cause pressure symptoms. Choked disc and neuritis optica has been noted only four times. (Braunstein, three times; Hölscher, once.) In acute cases a slight hyperemia of the background has been noted (Braunstein). Nystagmus, convergence, divergence of one eye, dilatation of the pupils have all been occasionally seen. In very rare cases where the extradural abscess is in the central fossa, crossed pareses may be observed, and Körner has noted sensory disturbances of speech in children in left-sided extradural abscess. In one case melancholia and in another the "idée fixe" have been described. (Biehl, Hölscher.)

If the abscess is located in the posterior cranial fossa, a stiff neck may be noted. In the majority of cases a high fever will be wanting, and if present we usually find an accompanying mastoid inflammation or a sinus phlebitis. If any change is noted it will be a lowering of the temperature. Usually the extradural, like the brain, abscess causes no change in the temperature curve. Headache is an inconstant symptom, at times it is entirely wanting and again it takes on the character of a brain abscess headache. It may be located over the ear, in the back of the head, or in other places which do not correspond to the location of the abscess, but is seldom diffuse in character. Mental or physical exertion or percussion over the seat of the abscess causes an increase in the headache, and it will usually be found to be worse at night.

Pain in the ear is often found in connection with the headache. Also as a frequent accompaniment of extradural abscess we have periostitis, formation of a fistulous opening and subperiosteal abscess. When it is remembered how seldom general symptoms and how still more rarely local symptoms are observed with extradural abscess, a proper differential diagnosis presents no difficulties. In brain abscess one will be able to recognize a more or less well marked symptom-complex, both general and local, while with an extradural abscess such a complex belongs to the exceptions. As has already been noted, a circumscribed bone change is usually found with the extradural abscess. An unusually abundant discharge of pus from the ear, which is quantitatively too great for a middle ear secretion, indicates an extradural abscess, and its presence is still more probable, if with the cessation of the discharge, the brain symptoms appear. If both diseases are present at the same time a correct diagnosis is difficult and often impossible, and if the extradural abscess runs its course with the symptoms of a brain abscess, errors are hard to avoid. In such cases the diagnosis is generally first made on the operation table.

The deep extradural abscess has the same course as the superficial, and often develops with the same symptoms or without any at all. The diagnosis of such an abscess is a remote possibility, and it is only after the mastoid has been opened and the cerebral and cerebellar abscesses excluded that we can arrive at a definite conclusion. A fistula into the labyrinth makes the diagnosis much easier.

An empyema of the saccus endolympathicus runs its course without symptoms and often leads to an abscess of the cerebellum.

The same conditons apply to extradural abscess and pachymeningitis interna, which also cause no typical symptoms with the exception of those cases where we have to deal with typical local symptoms due to softening of the brain cortex. In such cases a differential diagnosis between a brain abscess and a subdural or cortical abscess is impossible before the operation. Subdural abscesses can by reason of their extension and at times numerous complications, cause heterogenous and inexplicable symptoms. At times, local symptoms appear as a sensory aphasia, where the abscess is located in the left temporal lobe, so that the diagnosis lies between a meningitis and a temporal lobe abscess. In pachymeningitis and the encapsulated subdural abscess in the subarachnoid space, the spinal fluid is as a rule clear or only slightly clouded. When we find such a condition by means of a spinal punction we should think of an encapsulated collection of pus, so that a distinction between a subdural abscess and a brain abscess can not be made. If after incising the dura and the inner sinus wall no pus is discovered, the question arises whether we have to do with a brain abscess or a pachymeningitis interna without the formation of pus. (Hölcher.) The subdural abscess and the pachymeningitis may be the result of the breaking through of a brain abscess, but more often is a complication of a sinus thrombosus or an extradural abscess.

Encephalitis circumscripta and brain abscess are impossible to differentate in their earlier stages, as they both arise together, and as a matter of fact the encephalitis represents the initial stage of the abscess. In the differential diagnosis no great weight should be put on the duration of the disease, but the etiology above all must be taken into consideration.

In typical cases the differential diagnosis between brain abscess and leptomeningitis is not difficult to make. Purulent inflammation of the ear may be the etiologic basis for both diseases and both may have similar symptoms. Although as a rule each disease has a characteristic symptom complex, for itself, there are cases where one may be mistaken for the other and the difference is not therefore absolutely clear. When the leptomeningitis has a slow course it has a certain resemblance to a brain abscess. A headache which constantly reappears belongs to this group. In a few cases it is wanting or only poorly marked. Ultimately nausea, vomiting and dizziness are noted. Sensory disturbances are almost always present; they differ only in the method and time of their appearance. Their degree varies from a slight disturbance of the sensorium to the deepest coma. Under this class belong: excitement, irritability, uneasiness, sleeplessness, delirium, apathy, stupidity, etc., total or partial paralysis of the facialis, stiff neck (which is always well marked in meningitis), paralysis, myosis, sluggishness of the pupils, dread of light, later mydriasis, paralysis of the optic nerve, conjugate deviation, nystagmus, failure or increase in the patellar reflex, constipation and strangury. Macewen found neuritis and choked disc of rare occurrence, but Gowers considers them common accompaniments of basal meningitis. As is the case in a brain abscess patients are found in whom sensation is retained to the last, and the symptoms take on the appearance of an attack.

Körner noted aphasia when an exudate took place into the fossa of Sylvius. When the course of the meningitis is poorly marked the changes in temperature may be very slight or even subnormal or normal, and the pulse may also be lessened in frequency. With such a course the diagnosis presents almost insurmountable difficulties. As we have already shown, important data may be gained from a lumbar puncture. It is characteristic in leptomeningitis, that the cranial nerves are implicated to a much greater extent and more irregularly than is the case with a brain abscess. In meningitis the prodromal symptoms are of much longer duration. If the abscess lies in the region of the bridge, the cranial nerves may be widely affected, but as is well known, abscess in this region is exceedingly rare and, our therapeutic measures are without result. A false diagnosis in such a case is of no practical significance. Fever is never wanting in the acute cases and very often present in the subacute cases, its character varies, but is not at all typical. The temperature may go as high as 41°, C., but not usually over 39° C. Körner noted that a high fever predominates in meningitis of the convexity, and this type as a rule has a more severe course than basal meningitis. The pulse corresponds to the height of the temperature, and in the end stage is small, irregular and scarcely to be felt. In rare cases it remains strong and regular.

Hyperesthesia of the skin, clonic and tonic cramps of the facialis, and convulsions and paralysis of the opposite side have also been noted in leptomeningitis. The crossed paralyses involve equally the upper and lower extremities. In the terminal stage, we find convulsions and paralyses of the extremities, a partial or total loss of speech, and always progressive loss of consciousness, Kern's flexion contractures, boat shaped contracted abdomen, and involuntary passage of urine and feces after previous retention and constipation. The urine contains albumin, peptone and sugar. Local symptoms arising in the cranial nerves are usually characteristic of a basal meningitis, while symptoms in the extremities are the signs of a meningitis convexitatis. There is often a decided increase in the white blood cells, but this symptom has also been observed in an purulent inflammation of the mastoid, and a sinus thrombosis. Herpes of the lips and entrance to the nose often occur. The meningitis which comes on with the rupture of an abscess has an extremely rapid, stormy and apoplectic course and so conceals the symptoms of the abscess itself that the latter can be overlooked.

A sudden increase in the deafness of the sound side, is more significant of meningitis than brain abscess, although this symptom, as has already been stated, occurs in the latter.

In as much as the distal effects of the cerebellar abscess takes place along the meninges, the symptoms of both diseases, for that reason, may for a time be in doubt.

In children, a brain abscess of otitic origin and sinus phlebitis, often run their course with meningeal symptoms, and have often been mistaken for a meningitis. In other cases the abscess may occur with meningitis without exhibiting any characteristic symptoms, so that the effects of the latter alone are noted.

In many cases the existing symptoms may lead directly to a false diagnosis, in that either the presence of another complication with meningitis, or a meningitis coming in as a complication with another intracranial disease, may lead to a confusion as to the real state of affairs. Brieger, for example, observed a case of cerebellar abscess, whose symptoms reminded one strongly of meningitis, and the same thing has been seen with an extradural abscess.

The over-preponderance of the general symptoms in the clinical picture may give the impression of a general septic disease. We must also realize that an encapsulated collection of pus may give the appearance of a general meningitis, while on the other hand the distal effects of a meningitis may be confounded with a brain abscess.

Brain abscess and meningitis serosa are closely allied and often identical symptoms. The latter may exhibit the symptoms of a meningitis diffusa, but on the whole the prevailing symptom complex is that of pressure symptoms. A characteristic symptom as opposed to brain abscess is the appearance of neuritis optica, and an early profound loss of the sensations. Stiff neck, opisthotonos, differences in the size of the pupils, strabismus, headache, dizziness, vomiting, constipation, slowing of the pulse, temperature curve, and of the local symptoms, disturbances of speech, are common to both diseases. Disturbances of locomotion, which are so important in cerebellar abscesses, are still more marked in meningitis serosa. In the latter case disturbances of sight, such as amaurosis and amblyopia are very commonly observed. Deafness, loss of taste and smell, also double-sided pareses and paralyses of both legs, both arms, both facial nerves, both abducens nerves, are not infrequently met with in the case of meningitis serosa. At times these disturbances appear on one side only, and their subsequent involvement of the opposite side indicates a meningitis serosa as against a brain abscess. Koch has observed convulsive seizures in a third of the cases. Such attacks have been noted in a cerebellar abscess, but hydrocephalus was almost invariably an accompaniment. The principal difference lies in the course of the disease. The meningitis serosa develops suddenly with severe brain symptoms, which in a few days or even in a few hours entirely disappear without leaving any traces. The manifest and end stages of an abscess on the other hand, develop steadily onward toward the end, the symptoms lasting at the most a few weeks or only a few days, whereas the attacks of meningitis may be repeated throughout months or even years. The principal differential diagnostic point lies in the lumbar puncture. In general, the meningitis serosa is more often the complication of a brain abscess than it is of the ordinary purulent ear disease.

Tuberculous Meningitis and Brain Abscess.—It is to be especially noted that meningitis tuberculosa occurs especially in children under 10 years of age, and that brain abscess at this period is relatively uncommon. The disease has also been observed in adults up to thirty years of age. The meningitis is usually accompanied by general tuberculosis, but here and there one sees cases where the original trouble is lost sight of, and the meningitis appears as an apparently primary disease. Tubercular meningitis in children is usually accompanied by preliminary symptoms. They lose flesh, are weak, depressed, irritable, the sleep is uneasy, and they often complain of headache, which is increased by bodily work or mental exertion. Such a condition may be of weeks' duration. Then apparently without any couse, vomiting appears, which is usually accredited to an indiscretion in the diet and the headache becomes more violent. Symptoms of brain involvement appear which have the same character as a brain abscess, viz: somnolence, delirium, stiff neck, differences in the pupils, dizziness, convulsions, obstipation, and lowering of the pulse rate to 40 to the minute. The temperature increases a little, to 38.5° C. Local symptoms such as aphasia and poorly marked neuritis optica have been observed. The characteristic choroidal tubercles may be absent. At the end of the first or in the second week of the disease an involvement of the cranial nerve begins, shown by strabismus, inequality of the pupils, a slight degree of ptosis, and a paresis of the facialis. The somnolence passes into a coma, the head will be drawn backward, if this is not already the case, and a rigidity of the limbs may be noted. Commonly local convulsions or paralysis, hemiplegia, or paralysis of the arm or face, seldom of leg or face, will appear. The paralysis may be lasting or temporary. The pulse becomes rapid, 140 to 180, the respiration difficult and irregular, and the patient finally dies in coma. The temperature is either not elevated or is subnormal. At times just before death an apparent improvement takes place. If only the convexity of the brain is affected, which is seldom, the symptoms of cranial nerve involvement are wanting and vomiting is rare, and delirium, convulsions, and rigidity of the extremities become the prinpipal symptoms.

On account of the many symptoms common to these two diseases, and because of the possibility of an accidental tuberculosis in other organs, the difficulties of a differential diagnosis in a patient with a brain abscess of nontubercular origin, are often insurmountable. This is especially the case when one reflects that in children an acute middle ear inflammation is often seen with symptoms of headache, vomiting, fever, delirium, dizziness, strabismus, and a more or less well marked neuritis optica; and that the brain abscess often runs the course of a meningitis. In the best of cases, the diagnosis can only be probable.

If a child with no hereditary tuberculous tendencies is seized, during the course of an acute or middle ear inflammation, with attacks of severe head and ear pain, vomiting and other symptoms of intracranial pressure, with normal or subnormal temperature, with convulsions or loss of consciousness during which the mind remains clear, the symptom complex speaks for a brain abscess. In such cases stiffness of the muscles of the neck, pupil differences, fixation of the bulb, disturbances of equilibrium, and a staggering gait, indicate cerebellar abscess. A well marked neuritis optica at the end of the first week of sickness is also in favor of a cerebellar abscess. The diagnosis of a tubercular meningitis is made positive if the tuberculin reaction in the eye is positive, and if we find tubercle bacilli, lymphocytes, and a slight clouding in the spinal fluid. The tuberculin reaction and the presence of tubercle bacilli often fail. The puncture of the arachnoid sac during the mastoid operation may also be used as a diagnostic agent.

In large children and adults, the symptoms of a tubercular meningitis are the same with the exception that the prodromes and convulsions are rarer. As with abscess, the pulse and temperature remain the same. Sudden unconsciousness, delirium, marked stiffness of the neck muscles, and crossed disturbances of motion and sensation are more in favor of a meningitis, whose character, by an investigation of other organs and a careful review of the clinical history, may be more or less accurately ascertained. If tubercular caries of the middle ear and the adjacent parts are present, the meningitis is probability of tubercular origin. An abscess may, however, be present at the same time, but with the above mentioned general and local symptoms and a sound organ of hearing and with symptoms of tuberculosis in other parts of the body, a brain abscess is decidedly out of the question.

Virchow was the first to explain in what manner a collection of tubercles in the brain could lead to the formation of an abscess. Such a tubercle has its point of selection between the gray and the white matter. If the caseous contents begin to break down and liquefy, a thing which happens but seldom, we have in the middle of the tubercle a small space filled with a cloudy whey-like fluid. At times one notes that the tubercles are surrounded by pus or the entire mass is of a purulent nature. Such an abscess may be easily mistaken for a brain abscess of otitic origin, and is only to be differentiated by the presence of more or less tubercle bacilli. The symptoms are precisely similar to those of a brain abscess, but concomitant changes of a tubercular nature in the lungs or other bodily organs, will generally be found. The writer observed a brain abscess, with lung tuberculosis as a complication.

It would be difficult to make a mistake in the diagnosis between a brain abscess and meningitis cerebrospinalis epidemica. If we consider, in connection with the ear pain, the symptoms of the meningitis: the severe headache, retraction of the head, cutaneous hyperesthesia, delirium, pain in the back, muscular rigidity, pain in the neck, muscles, herpes labialis, even leaving out of consideration the epidemic and infectious nature of the disease, an error as to the real nature of the trouble is almost impossible.

Sinus Thrombosis and Brain Abscess.—A confounding of these two troubles is uncommon. In most cases, at least in the beginning, headache and vomiting will accompany the thrombosis. The headache is at times diffuse at times sharply circumscribed on the diseased side or referred to the ear itself. In cases where the condition is not complicated with a brain abscess or meningitis, the consciousness is not at first disturbed, but later it may be clouded to a slight degree. The opposite of these conditions has been noted in scattered cases. The author observed several cases of septic thrombus where the autopsy showed a well marked basal meningitis, in which not taking into account the stupor depending upon the infectious process itself, the consciousness was not at all disturbed during life, but only a few hours just before death. Psychical depression, a slight degree of stupor and a general loss of strength are very common. Neuritis optica is seldom observed. Körner has never observed it in cases of the transverse sinus, but it does appear in diseases of the cavernous sinus, and Jansen considers it here of diagnostic value.

The most important differential symptom which separates an infected sinus thrombosis from a brain abscess, is the characteristic septic fever, with its accompanying chills, sweating and rapid rises and falls in the temperature (41.5° to 36.2°). In children, the chills and sweating may be wanting, and the fever itself, especially when metastases are present, may not show the characteristic curve. The pulse corresponds to the temperature, and when a sepsis is present, the acceleration will be noted during the afebrile intervals. A slowing of the pulse to 42 has been noted in rare cases. (Kessel.)

Metastatic brain abscesses are very rare and usually multiple. A single abscess only may be present, which is therefore operable. The metastatic abscesses originating in a purulent ear discharge, are usually peripheral, and occur especially in the joints, subcutaneous tissues and muscles, seldom in the lungs, and rarely in other organs.

External manifestations which characterize a phlebothrombosis, are involvement of the nerves which pass through the foramen jugulare (vagus, accessorius, glossopharyngeus), as well as the hypoglossus. In addition we may find a painful edema of the posterior border of the mastoid, edema of the eyelids (in diseases of the sinus cavernous), a marked filling of the veins of the forehead on the diseased side and unequal fullness of the external jugulars. Often a hard painful cordlike swelling of the thrombosed vein and the infiltrated connective tissue may be distinctly felt. Movement or turning the head toward the sound side causes pain. Hoarseness, aphonia and difficulty of breathing are often present. Localized convulsions in the region supplied by the accessorius, paralysis of the muscles involved in the act of swallowing, paralyses of the muscles of the soft palate, all come into consideration.

Plugging of the sinus cavernosus makes itself known through a passive hyperemia in the region of the ophthalmic vein and the disturbances of one or more nerves which stand in close relation to it. A high continuous fever, a weak and rapid pulse, disturbances of consciousness and a marked loss of strength make up the symptoms of a septic intoxication.

In children an uncomplicated sinus thrombus can, at times, run its course with the so-called meningeal symptoms. In such a case a certain diagnosis is not to be made, but a lumbar puncture will help to clear the matter up.

If a sinus thrombosis is complicated with a brain abscess, or if at the same time a leptomeningitis is present, the symptoms of the general infection are so well marked that the symptoms of the complicating disease are to a certain extent clouded. In many such cases the brain abscess is small and develops after the formation of the thrombus. (Macewen.)

Brain Abscess and Apoplexy. — The end stage of the brain abscess or its final rupture into the side ventricle or upon the surface of the brain, has many points of similiarity with an attack of apoplexy. This is especially true if the etiologic cause of the brain abscess has been disregarded. On the other hand, apoplexy, when accompanied by a purulent middle ear disease, may be mistaken for an abscess. In both diseases two main sets of symptoms dominate: general and local. The attack can be accompanied by preliminary symptoms, such as headache, dizziness, slight physical disturbances, slight disturbances of speech, which with the presence of a purulent ear discharge cause one to be suspicious of a latent running or ruptured abscess. Apoplexy, however, comes in the latter periods of life, while abscess is usually seen during the first three decenniums. In apoplexy, of the general symptoms, loss of consciousness, and the local hemiplegia, are the most common symptoms. The apoplectic attack comes on suddenly, the patient collapses into a comatose condition, and for a shorter or longer time is wholly or entirely unconscious. Movements and sensations are entirely blotted out, and the patient may die in a few hours or even a few minutes. The pulse is at first slowed, small and hard to feel. At times it is accelerated. About an hour after an attack the temperature sinks as low as 30° and remains so until death or rises again to normal. A convulsion may accompany the attack, but it is as a rule wanting. In certain cases the patient may recover, the general symptoms recede and the hemiplegia alone remain. The rupture of an abscess will almost always be accompanied by a chill, increased temperature, violent headaches and vomiting. The pulse is greatly accelerated, and the other symptoms, already mentioned, may appear. The attack is invariably fatal. As a rule it can be ascertained that with the brain abscess, certain general symptoms, and often local symptoms have been present for a longer or shorter period, while in apoplectic cases these are generally wanting or when they did appear were of short duration and not characteristic.

Brain Abscess and Tumor.—Here the etiology is of great importance. The very absence of a specific etiologic factor would speak for a tumor. Distinct local brain symptoms and a slow gradual increase in the course of the same, especially with paralysis of the cranial nerves, a high degree of neuritis optica, with often progressive blindness, at times hemianopsia, general and local convulsions, general muscular weakness, attacks of loss of consciousness, all favor a new growth. The receding of severe symptoms also indicates a tumor. The temperature never vacillates, but as we have already shown the course of the abscess can be an afebrile one or the curve may even drop below the normal. In the latter affection, however, there will be slight changes in the temperature with generally an increase toward the end, following the appearance of complications.

The rapid development of acute or severe brain symptoms after only slight signs of a brain affection have been noted, indicates an abscess. The symptoms common to both troubles are,—headache, dizziness, vomiting, stupor of the mental faculties, neuritis optica, uncommon in abscess and not so well marked as in tumor, and lastly cerebellar ataxia in the case of cerebellar tumors. Neuritis optica is sometimes a very early symptom, especially in tumors of the cerebellum and diseases of the copora quadrigemina. In tumors of the cerebrum it makes its appearance late.

Convulsions often appear in the case of a tumor before a suspicion of the growth is present. Although in the case of a tumor, the development of the symptoms is slow and gradual, the course of the individual symptoms is not always equally so. In slow growths, as is the case with an abscess, the process of the development may be intermittent or even stationary. Pressure symptoms may appear quickly in the case of a tumor; a healthy facial nerve, for example, can in a few days be completely paralyzed. In other cases a rapid increase in the symptoms occur as a result of a local meningitis caused by the tumor. Commonly there follows a temporary or lasting hemiplegia, following a convulsion, or even without one. In the cases of recognizable syphilis, it is probable that the tumor is of this nature, and eventually it forms a gummi which under certain circumstances may break down and in this manner give an indistinct picture of a brain abscess. The diagnosis, as is the case in the tuberculous, may be only an indefinite one.

The intracranial aneurysm also forms a new growth and gives rise to symptoms of a tumor of an entirely different nature. The only difference is the presence within the cranium of an audible aneurysmal murmur.

In very rare cases a new growth can take place in the presence of a purulent ear disease, and this may cause the diagnosis to become a matter of great difficulty or even impossibility.

Hysteria may be mistaken for an abscess, when it occurs in connection with an ear discharge or a purulent affection of the temporal bone. This is especially the case if the patient has been under observation for a short time, and if headache and vomiting are present. The sudden outbreak, the failure of all symptoms of organic disease or neuritis optica, the method and appearance of the objective symptoms, as well as the circumstance that hysterical individuals have a tendency to imitate allows one to make the correct diagnosis. Hemianesthesia, disturbances of the special senses without a concomitant motor paralysis, are among the most infrequent of the symptoms observed in brain abscesses or tumors.

In females, and perhaps where other hysterical symptoms are present, it sometimes happens that a brain abscess is mistaken for hysteria. In order to exclude diagnostic errors as far as possible, the apparent hysterical symptoms must never be allowed to influence the diagnosis, until an organic brain affection can be positively excluded. The best protection against diagnostic mistakes is an exact knowledge of hysteria, and an exact knowledge of the brain abscess, as well as a rich experience in these fields. Upon the other hand, it must not be forgotten that the diagnosis of hysteria presents, in most cases, no especial difficulty if we take into account the manner and origin of the disease, the course of the same, and the presence of certain characteristic symptoms. Experience has taught us, however, that hysteria may give rise to diagnostic errors, and to determine whether certain symptoms belong to the hysteria or not, presents problems which are difficult even for the experienced observer to solve. If it is necessary in a given case to make a positive diagnosis, the principal thing to do is to seek for the appearance of the stigmata of the disease. These consist of psychic and somatic disturbances, anesthesias, hysterical zones, attacks of convulsions, etc. In seeking for stigmata, organic lesions, especially those characteristic for brain abscess, must be excluded. It is to be regretted that many symptoms which are characteristic for brain abscess are often found in hysteria.,

With the exception of certain symptoms, the method of origin and the further development of the disease, allow one to come to a fairly definite conclusion as to its nature. In hysteria the course of the disease is very seldom regular. Severe disturbances may suddenly disappear without improvement of the general condition. Attacks which suddenly develop with great violence, as well as a sudden cessation of long standing symptoms, point to hysteria. A number of hysterical symptoms, such as anesthesias, hyperesthesias, paretic conditions, contractions, tremors, etc., may suddenly run from one side of the body to the other. The grouping together of certain symptoms often gives us certain diagnostic starting points, as for example: the hysterical hemianesthesias do not occur with hemiopia, but the hysterical paraplegias do occur with anesthesias which are confined to the legs.

An example from von Boissard serves to show how easily hysteria can be mistaken for a severe brain complication. A female patient, 45 years of age, was suddently attacked with severe headache and vomiting of a cerebral character. The pupils were small, the pulse slowed to 48, and obstipation was present. The temperature was normal. As a member of the family had died of pulmonary tuberculosis, the suspicion pointed to meningitis tuberculosa. After a few days the condition improved, a right sided hemianesthesia appeared, and immediately the hysterical nature of the trouble was apparent.

The author, a few years ago observed the case of a young man of 26, upon whom a mastoid operation was made, following a right sided chronic ear discharge. The patient complained continually of symptoms that pointed to a brain abscess. At the hands of a surgeon he was trephined twice without result, and the brain substance explored. The entire condition was that of a hysterical person who rather expected further operations on the skull.

Neurasthenia, especially when an ear discharge is present, may often give rise to the suspicion of a brain abscess. This latter disease may often closely simulate the cerebral type of neurasthenia, especially in its preliminary period. In the further course of the trouble, there comes with these doubtful symptoms, those which can only be explained as depending upon an anatomic change. Just in proportion as the symptoms depending upon an anatomic basis are not present, just in that proportion must, *ceteris paribus*, neurasthenia be taken into consideration. It is not correct to say that symptoms of neurasthenia are of a fluctuating character. In many cases they are very obstinate, especially dullness in the head, the nervous asthenopia, the decrease in the ability for mental work, weakness in the legs, etc., symptoms which can not be looked upon as harmless as they are often seen in brain abscess. On the other hand, at least a certain proportion of the brain abscess symptoms show a marked degree of fluctuation. In neurasthenics, the headache seldom reaches a great intensity without special cause, and less seldom continues such intensity over a long period of time. Percussion tenderness of the skull and dizziness are not present. Vomiting, a symptom which is always present with brain abscess, is always absent in neurasthenia. Mental depression, remarkable changes in the voice, transient changes in the strength, and difference in the size of the pupils, are common to both diseases.

Hyperesthesia of the scalp is common with neurasthenia, but rare in brain abscess. Aphasic changes in speech are wanting in neurasthenia, but in rare cases a paraphasia, word amnesia, or paragraphia may be present. Of the motor disturbances, a long continuing paralysis of the extremities fails absolutely. Paralytic disturbances of the facialis, and of the eye muscles, myosis, reflex immobility of the pupils, changing reaction to light, slight pareses in the region of the above mentioned nerves, must be considered as all speaking against neurasthenia. On the average the pulse is 80 to 100. In neurasthenia, all changes of hearing, tuning fork tests and bone conduction must be considered of labyrinthine nature, and, according to Babinsky, of central origin. They are only noted in traumatic neurasthenia.

Exceptionally, coma diabeticum may, in a person suffering from an ear discharge, bear a striking resemblance to a brain abscess in the end stage, and thus give rise to diagnostic errors. This is apt to be the case, if the attack comes on suddenly, and if the patient has been seen for the first time. The patient is entirely unconscious; the coma is seldom broken by periods of consciousness. Occasionally unmistakable clonic convulsions appear, and mydriasis, fixation of the pupil, half opened lids, small and quick pulse, temperature below normal, deep inspiration with short expiration, and growing cyanosis, are observed. The history usually shows previous headache, loss of sleep, uneasiness, anxiety, dizziness, and a continuous feeling of intoxication. The penetrating odor of acetone, directs us to the proper differential diagnosis. A further diagnostic point is the presence of sugar in the urine and its exceptionally strong reaction to chlorid of iron. Naturally a diabetic coma and a brain abscess may be present at the same time, but under these circumstances an inability to make a correct differential diagnosis is of little practical value.

Uremia, occurring together with a purulent otitis, may give occasion to a confusion of diagnosis. The first attacks, which make themselves known by loss of consciousness, coma, and tonic or clonic convulsions, are usually preceded by headache, dizziness, apathy, a slight dulling of the sensations, an increased desire for sleep, and not infrequently nausea, vomiting and diminished pulse rate. With an abnormally high and at other times an abnormally low temperature, 42°-30° C. paralyses, contractures, and twitching of the muscles, are but seldom observed and still more infrequently mania, melancholic depression, and delirium. A consideration of the reasons for the suppressed or decreased secretion of the urine, as well as an investigation of the fundi for retinitis albuminurica, will almost always guard against a mistake in diagnosis. The amaurosis, which not infrequently develops without any anatomic ground, demonstrable with the opthalmoscope, and which may also completely disappear in a short time, is a condition never seen in brain abscess. Here also, in connection with, but not depending on the disease which gave rise to the brain abscess, we may have a brain abscess, and the diagnosis may of course present great difficulties, and often make it almost impossible to arrive at the correct solution.

The sequence of the symptoms in typhoid fever is so peculiarly characteristic, that a mistake can scarcely be made in its recognition. However, this regularity in sequence, especially in the temperature curve, can not always be expected, and the patient must be under observation for a certain period of time. It is easier to confound typhoid with pyemia than with brain abscess. We must never lose sight of the fact, that we may have a typhoid with a low temperature, slowed pulse and preponderating brain symptoms, and thus give occasion to an error. An exact knowledge of both diseases, a continuous and accurate observation of the course of the disease and the agglutination will be enough to protect one from all diagnostic failures.

Up to the present writing the writer has been twice consulted by internists, where the suspicion of the brain abscess was present. An exact consideration and investigation of the symptoms convinced him that he had to do with a typhoid fever. In the one case there was no ear discharge present, the physician basing his diagnosis on circumscribed headache, percussion pains, boring pains in the ear, torpid mentality, vomiting and heightened fever.

Epilepsy is not difficult to differentiate from brain abscess. The epileptic attack is characterized by sudden entire loss of consciousness, sudden falling to the ground, and a well marked facial pallor which rapidly changes to a red or cyanotic color. It is very often accompanied by a piercing cry and rigidity of the body, and tonic and clonic convulsions quickly appear. An entire loss of sensation and every psychic process is observed. The pupils are dilated, and do not react to light, trismus, opisthotonos, a strongly beating heart, and blood flecked lips, are all to be noted. After a longer or shorter period, from 10 to 15 minutes, but at most an hour, the attack ceases. Epilepsy occurs usually before the twentieth year, later it is of rare occurrence. Loss of consciousness and convulsions are also noted in abscess and the attack can also disappear but neuritis optica will be present, the convulsions will not be so general and will be accompanied by paralysis.

It is possible that a confusion could arise between brain abscess and intermittent fever. In any event an uncertainty in such cases could last for a short time only, as for instance in rare cases of malaria, which have a course without fever or enlargement of the spleen. The high temperature, the continuous chills preceded by a feeling of heat, and the examination of the blood, would all guard against a mistake in diagnosis. As is well known, chills and fever, during the manifest stage of the brain abscess are practically unknown and can occur only in the prodromal and end periods. In the latter case, death invariably follows, while a death during an attack of chills and fever with malaria is practically unknown.

As a curiosity, the writer was twice consulted in cases of circumscribed and rheumatic pain in the region of the temporal bone, which came on in paroxysms and in the absence of any ear disease, gave rise to a suspicion of brain abscess.

From all that has been said, the diagnosis of brain abscess can be made on the average with a certain degree of certainty, but here as in all other branches of medicine, cases occur which are contradictory, not well marked, and difficult to make clear. When errors of diagnosis are made, or when the abscess is not recognized, then the cause lies in the disease itself or in the similarity of the remaining endocranial complications, which depend upon a purulent process arising in the organ of hearing. In addition many of these complications make their appearance at the same time with the abscess, or are caused by the same, and therefore cloud the picture of the abscess itself. Finally we must take into consideration the similarity of the symptoms of the brain abscess, with other general diseases where no pus process is present.

It is to be hoped, that with the progress of science, the methods of determining the brain abscess will also be perfected. It is doubtful, whether in the future, a brain abscess can always with certainty be diagnosed, for errors will always be a part of man's nature, and to that end we must strive as fas as possible to eliminate them by exact observation, rich experience and correct judgment.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINOLOGIC AND LARYNGOLOGIC LITERATURE.

I—EAR.

The Treatment and Prognosis of Chronic Middle Ear Discharge.

RUPPERT, Munich (*Münch. med. Wochenschr.,* No. 21, 1908). This paper, appearing as it did just before the meeting of the German Otological Congress in June, 1908, was very favorably received there, voicing as it did the sentiments of the famous head of the Munich Clinic, Prof. Bezold, where conservative treatment of middle ear suppurations is carried to its extreme limits and operative interference is the exception. The author, one of the assistants in the klinik, recognizes the fallibility of statistics based on ambulatory clinical material, where the attendance is always irregular and where the assistants are always changing, and takes instead the statistics from Dr. Schiebe of Munich, a former pupil of Prof. Bezold. In the seventeen years covered in the investigation, from 1889 to 1906, the author acted as assistant for the last four. Bezold's boric acid powder treatment was the only method used. The patients were treated every day until the abatement of the discharge, then two to three times a week. Under the term "Chronic middle ear discharge" the author places only those cases of inflammation of the middle ear cavities where a perforation remains continuously open and discharges for years, following the original acute attack, or where the perforation has remained opened because of scar tissue and after years sets up a chronic discharge. Even cases of acute middle ear suppuration which continues a full year before resolution, were not considered in these statistics, as the perforation finally closed, when the discharged ceased. In the seventeen years 1,118 cases of chronic middle ear discharge were treated; of these 38 or 3.4 per cent showed complications from the beginning. The remaining 1,080 were uncomplicated. Of this number 592 or 55 per cent had a central perforation: the remaining were either in Schrapnell's membrane or involved the annulus tympanicus. The minute description of the technic of the treatment, which because it is a type of this method as it is so commonly carried out here in Germany, is repeated here in detail. After a thorough syringing out of the external auditory meatus with a 4 per cent solution of boric acid and Politzerization, a fine, curved cotton applicator was used very carefully to dry out the middle ear by introducing the cotton directly through the perforation. An insufflation of the most finely powdered boric acid was then made.

In 10.1 per cent of the cases with central perforation the formation of granulations and polypi was noted. These were removed only with the snare or curette; the author claiming a certain danger of facial paresis or even total deafness through the use of the chemical cauterants.

The disagreeable odor disappeared after the first treatment or at latest after the second. Three hundred and seventy-one cases or 93.9 per cent were completely relieved for a period of always more than a half year following the cessation of the treatment. In 18.5 per cent the discharge ceased after the first treatment. In 28.1 per cent the discharge ceased inside of a week. In 28.6 per cent the discharge ceased inside of a month. In 8.6 per cent the discharge ceased inside of two months. In 3.5 per cent the discharge ceased inside of three months. In 3.5 per cent the discharge ceased inside of six months. In only 2 per cent did the treatment last a year, and in four cases or 1 per cent the treatment was carried out for a longer period of time. In 24 cases or 6.1 per cent the discharge continued in spite of treatment covering years. An interesting analysis of the cases where the healing covered a period exceeding six months is given. Some of the cases were treated at home, others lived under very unfavorable conditions and in still others a strumous diathesis seemed to play an important role in the delay.

Most of the 124 uncured cases have been under observation from 5 to 18 years and none has developed complications or shown a tendency to recurrence of the fetid secretion. One of the cases which had been so long in treatment was operated on (not by the author), but the discharge never ceased.

Of the 395 central perforations two were operated upon by the author. In one with a small round central perforation which after more than six months' treatment, discharged a strongly smelling crumbly pus, the radical operation was performed. No cholesteatoma or necrosis was found and the author believes that a little more patience would have led to a complete conservative healing.

The second case was a hysterical individual, whose alarming symptoms pointed to a meningitis serosa and involvement of the labyrinth. Nothing but a diseased condition of the mucous membrane was found at the operation.

In seventeen years 489 cases of perforations involving the annulus were observed. Of these, 359 remained under treatment. Here anatomically and pathologically a new set of conditions was to be combated. The Hartmann's tympanic tubes were used to wash out the attic and antrum; a drying with cotton on the end of a carrier with a slight bend upward, followed, and the treatment was concluded by blowing powdered boric acid through the perforation into the antrum itself.

The formation of polypi was much more common in this group, 39.8 per cent as compared with the central perforation cases. The extraction of the ossicles was not found necessary in a single instance. The author saw many cases of so-called caries, with cholesteatomous masses in the antrum go to complete healing after the conservative treatment of the discharge!

Of these 359 cases, 82 were perforations in Schrapnell's membrane. The formation of cholesteatoma was noted in 53.8 per cent of the perforations which involved the margo tympanicus and in 70.5 per cent of the cases with the perforation in Schrapnell's membrane.

As was to be expected, the time of healing in this group was notably longer than in the first: 8.9 per cent were cured by the first treatment; 18.4 per cent inside of eight days; 25.6 per cent within one month; 11.7 per cent within two months; 5.6 per cent within three months; 7.6 per cent within six months; 7.2 per cent within twelve months; 4.5 per cent more than a year; 10.6 per cent remained uncured. In the 359 cases, 357 were healed by conservative methods and 7 came to operation. The remarkable results achieved here and the general discussion of the same theme later in the German Otological Congress, gives one a good idea of the conservative methods now in vogue in Germany. *Horn.*

A Case of Mastoiditis with Brain Complications.

Knause, New York (*Archives of Otology*, Vol. xxxvii, No 2). A young man gave a history of earache lasting one week. In spite of a paracentesis mastoid tenderness persisted and edema developed over the mastoid and extended backward toward the occiput.

On operation a subperiosteal abscess was drained, a perforation was found in the region of the zygomatic cells and another over the tip. His condition was unimproved. On the third day after operation he was restless, at times delirious, temperature 103° F., pulse 104,, respiration 24. There were tenderness over and rigidity of the posterior cervical muscles. Beginning choked disc on the left side. Cerebration was slow and he had agraphic aphasia. On lumbar puncture the fluid came slowly, was slightly turbid and free from bacteria.

Seven days after the mastoid operation he had a tonic convulsion with marked retraction of the head, this was followed by a paralysis of the lower branches of the right facial nerve. Next day rectal temperature was 97.8° F., pulse 54, respiration 14, and an exploration was made for brain abscess. The incision for exposure of the mastoid was carried forward and upward for about two inches and after exposure of the dura a narrow blade was passed in various directions but without result. His general condition was much improved after this brain exploration, then he contracted erysipelas; from this he was convalescing nicely, being even able to be out of doors, when he suddenly died. On autopsy the dura was found very adherent to the left temporosphenoidal lobe. This lobe was yellowish-green in color, soft and mushy and on section about half an ounce of greenish pus escaped.

There was acute parenchymatous degeneration of the heart muscle, acute miliary tuberculosis of the lungs, acute tubercular splenitis, acute parenchymatous nephritis and fatty cirrhosis of the liver. The cerebral abscess was opposite the kidney-shaped opening made in the skull and its wall was not more than one-half inch in thickness. *Campbell.*

Report of Two Cases of Sinus Thrombosis Complicated by Cerebral Abscess in the Temporo-Sphenoidal Lobes.

DABNEY, Marietta, Ohio (*Archives of Otology*, Vol. xxxvii, No. 2). CASE 1. A man, aged 48; one month before coming under observation developed an acute suppurative otitis media of the left ear. There has been continuous pain in the ear, over the mastoid and more or less pain of the entire head There is mental hebetude. Temperature 101° F. and the ear discharge is blood-streaked. Smears show streptococci. Drainage appears to be insufficient. There is sagging of the posterior-superior wall and swelling of the tissues over the mastoid. On operation pus was found in the antrum and granulation tissue filled the bony trabeculae. On clearing out the granulations in the tympanic cavity there was found an erosion in the roof of the tympanum, but the dura seemed to be intact.

On the third day after operation he felt very well after the dressings were changed. Babinski reflex slight, Kerning's sign positive. The pulse, however, was rapid. On the fifth day he could not be roused, both pupils were dilated and there was motor paralysis over the entire right side of the body. Brain abscess was diagnosed and located, probably, in the region of the lenticular nucleus, where the motor tracts cross.

Under anesthesia the perforation in the roof of the tympanum was enlarged and the dura incised outward and backward along the floor of the middle fossa. A grooved director was then passed upward and inward into the brain substance for a distance of one and one-fourth inches and liberated about two drams of fetid pus. This pus contained streptococci. Two days later the patient died without having regained consciousness. Autopsy was not permitted.

Case 2. A boy, aged 15, had had a number of attacks of purulent otitis in the right ear. The present attack began ten days ago. He has the appearance of one suffering from septicemia. Temperature 103° F. There is edema over the mastoid and the slightest pressure there causes excruciating pain. The posterior-superior canal wall sags and the sanguinolent pus contains streptococci and Weichselbaum's diplococci.

The entire mastoid structure was found filled with the products of inflammation. The antrum was filled with cholesteatoma and granulation tissue protruded from an erosion in the roof of the antrum. While curetting this fistulous opening a small subdural abscess was opened and drained. The dural opening was enlarged and an area of necrotic brain tissue about the size of a large hazelnut was found and removed.

During the next 24 hours the patient had two chills and sweated profusedly. The temperature ranged from 100° F. to 105° F. and pulse 50 to 106. Along the course of the internal jugular vein the neck became very tender and edematous, and he developed a cough with prune-juice expectoration.

Sigmoid sinus thrombosis was suspected. The internal jugular vein from the clavicle to the skull was resected. The sinus was uncovered and its parietal wall was found thickened and covered with a layer of plastic lymph. As the sinus was about to be opened the patient failed so rapidly that operation was discontinued.

For the next two days he was rational at times, then this would be followed by maniacal delirium. The pupils were dilated There was convergent strabismus of the right eye, rigidity of the neck and slight retraction of the head. Motor paralysis of the left arm and leg with ankle clonus well marked on this side. He is very irritable and replies slowly when questioned. Temperature 100° F. to 102° F., pulse 45 to 56. It was suspected that another cranial abscess was forming. The mastoid wound was extended upward and forward for three-fourths of an inch above the temporal ridge, the dura was incised and a bistoury carried upward and inward into the brain substance until the abscess was reached. The sinus was also opened and a small elastic clot found near the jugular bulb.

From the time of opening the second abscess his recovery was speedy. *Campbell.*

A Case of Acute Internal Hydrocephalus Secondary to Streptococcal Infection of the Labyrinth.

SCOTT, London, Eng. (*Archives of Otology,* Vol. xxxvii, No. 2). A boy, aged 14, had a radical mastoid operation performed on account of an uncomplicated chronic suppuration of the left middle ear. There developed an acute internal hydrocephalus secondary to infection of the labyrinth and unattended by gross lesions such as brain abscess or meningitis.

Clinical tests for hearing were unreliable because the boy was mentally backward. Vomiting was the first untoward symptom; this occurred every day for five days after the operation. This was due, as autopsy revealed, to infection of the labyrinth. There must have been giddiness. Yet, of this, he made no complaint.

Severe headache masked all symptoms and it was produced by the increasing distention of the ventricles. Had the labyrinth been drained and at the same time the distended ventricles been relieved by lumbar puncture or puncture of the brain, life might have been saved. *Campbell.*

Report of a Case of Meningitis of Otitic Origin; Operation; Recovery.

KERRISON, New York (*Archives of Otology,* Vol. xxxvii, No. 2). A man, aged 34, with a fetid otitis media suppurativa which had persisted since childhood suddenly developed a severe left earache accompanied by high fever, severe occipital pains and vomiting. His mental condition was dull, leucocyte count 22,800 and polymorpho-nucleophiles 88 per cent. Unconsciousness developed, with contracted pupils and rigidity of the muscles of the back of the neck.

A mastoid operation was done and the incision carried upward so as to expose the middle fossa and temporo-sphenoidal lobe. The dura bulged, was congested and it was incised Considerable oozing of cerebral fluid followed. A grooved director was passed in different directions into the brain substance. After being passed backward and inward for about two and three-fourths inches, there followed a free flow of cerebral fluid. The lateral ventricle had been entered. This completed the operation and gauze drainage kept the operative wound open for six weeks, while the cerebro-spinal fluid continued to flow. For the first 24 hours morphin was needed to allay the pain and after this the course was favorable.

Campbell.

The "Piano-String" Theory of the Basilar Membrane.

BRYANT, New York (*Archives of Otology,* Vol. xxxvii. No. 2). The author's conclusions are:

1. The basilar membrane is not essential to the organ of Corti, and when present is not furnished with the requisite length and mass of fibres to vibrate in sympathy with every note, even if the rest of the structures would allow it.

2. The basilar membrane is devoid of the requirements of a resonating body.

3. Helmholtz's "piano-string" theory of musical perception is without foundation in every particular. *Campbell.*

Sarcoma of the Middle Ear.

ZEBROWSKI, Warsaw (*Archives of Otology,* Vol. xxxvii, No. 2). The number of cases recorded is less than fifty. The author's is that of a man, aged 25, who has had a tumor growth in and about the left ear for eight months. There was total paralysis of the left facial nerve.

On operation the tumor mass was found to occupy the middle ear including the antrum and mastoid cells and also a space extending from the mastoid tip to the transverse process of the atlas. These soft gray masses were easily removed by a sharp spoon, but the growth promptly recurred and one week later a tumor the size of a pigeon's egg was removed from the left shin bone which on microscopic examination proved to be a round cell sarcoma.

On postmortem examination it was found that the growth filled the nasopharynx, had in part destroyed the sphenoid bone and joints of the occipital bone. The odontoid process had entered the skull cavity and destroyed the medulla. There were metastases in the lungs and heart muscle. The pericardium contained about 1000 cc. of reddish muddy fluid. The labyrinth was uninvolved. An interesting point was noted in this case as regards facial palsy in the differential diagnosis between primary and secondary sarcoma of the middle ear. This paralysis occurs only in cases of primary middle-ear sarcoma. *Campbell.*

A Case of Large Cholesteatoma of the Middle Ear and the Posterior Cranial Fossa, Cured by Radical Operation. No Recurrence After Eight and One-half Years.

ZIMMERMANN, Milwaukee (*Archives of Otology,* Vol. xxxvii, No. 2). A man, aged 36, in whose left ear polypoid masses projected from the external meatus. The middle ear was extensively necrosed. From the right ear a polyp and fetid cholesteatoma were removed. Eighteen months later an abscess formed and was opened on the right side of the neck one and one-half inches below the mastoid tip. This abscess cavity ran down along the sterno-cleido-mastoid muscle (Bezold's mastoiditis). The right facial nerve was paralyzed, there was a fetid otorrhea, the posterior-superior canal bulged and cholesteatomatous masses were removed from the attic. A radical mastoid operation was performed; when the cortex was removed a large cavity was found filled with a fetid cholesteatoma and when this was removed the lateral sinus and thickened dura was exposed. The medial portion of the posterior meatal wall formed a loose sequester.

Recovery was perfect and the facial paralysis partially cured. The eye can be closed completely but the angle of the mouth droops slightly. A permanent post-auricular opening was left.

Campbell.

II—NOSE.

Nasal Headache as a Result of Hyperemia of the Central Nervous Organs.

Rethi (*Mediz. Klinik.*, No. 16, 1908) is doubtful of such a hyperemia as a general rule. He believes, however, that certain high degrees of nasal hypertrophy may cause severe headaches, but also says that the disappearance of the headache, after the application of cocain in the nose, is not caused by the astringent action, which theoretically would cause its increase, but due only to its local anesthetic action. *Horn.*

The Connection Between Diseases of the Stomach and the Nose.

Hecht (*Münch. med. Wochenschr.*, No. 12, 1908) shows by quoting two cases of empyema of the antrum of Highmore, how a continuous swallowing of pus, year after year, may set up a stomach catarrh of severe character, with loss of flesh, nausea and vomiting. All treatment by internists was without avail until a specialist discovered the source of trouble. The antrum was washed out and an amelioration of the stomach symptoms immediately followed. He deprecates the fact that so little weight is laid upon this connection in text books of diseases of the stomach. *Horn.*

The Treatment of Ozena by Means of Gargles.

Schmidt (*Münch. med. Wochenschr.*, No. 23, 1908) recommends gargling with closed mouth in cases where the crusts are impacted in the nasopharynx, and can not be loosened by nasal irrigation. A part of the water is forced behind the soft palate and the crusts are thereby loosened.

Horn.

The Nasal Reflexes.

Koblanck (*Deutch. med. Wochenschr.*, No. 24, 1908). In a most original and stimulating article, covered in the above heading, Koblanck quotes some remarkable cases, where a simple caustic of the inferior turbinate or the application of cocain to the nasal mucous membrane, has caused a marked improvement of various disorders connected with the sexual organs. During childbirth, the application of a 20 per cent cocain solution has often favorably influenced the pains and increased the strength of the uterine contractions. In dysmenorrhea he considers it a most valuable diagnostic and therapeutic help. In twelve women who had always been sterile and where no cause could be found, a nasal treatment resulted in 9 per cent of them becoming pregnant, all within a period of a few months! In perityphlitis fálsa he found it invaluable in differential diagnosis. In two cases of sciatica he secured a complete cure. In several cases of sexual hyperesthesia a cure also resulted as a result of local caustic operation. The results are not to be explained by suggestion and the various sudden deaths which have followed simple nasal operations are not due to the chloroform or cocain but to this intimate relation between the intumescent body and the central nervous system. *Horn.*

Nasal Dysmenorrhea.

KUTTNER (*Deut. med. Wochenschr.*, No. 24, 1908). Almost in direct opposition to the findings of Koblanck, contained in the article just reviewed, Kuttner says that "the center (standing in direct relation with the genital apparatus of woman) does not and could not exist." Although he has seen many cases of dysmenorrhea favorably influenced and even cured by local nasal applications he places it largely to the credit of suggestion, the general influence of the cocain, or the general well-being after the nasal trouble is corrected. Literature accompanies the article. *Horn.*

III—LARYNX.

The Pathology of Laryngocele.

SCHEVEN, Frankfurt (*Münch. med. Wochenschr.*, No. 9, 1908), recites one new case and reviews literature. *Horn.*

Clinical Notes on Laryngeal Tuberculosis.

BEVERLEY ROBINSON (*Am. Jour. Med. Sci.*, August, 1908). In the way of protective and curative treatment, two things are necessary: (1) rest for the larynx, and (2) inhalations. Personal experiences since 1885 has proved to the author the great value of the frequent, persistent, long-continued use of the perforated zinc inhaler with inhalations of creosote and alcohol, or, when there is much irritative cough, of beechwood creosote, alcohol, and spirit of chloroform in equal parts.

Richards.

IV—PHARYNX.

A Hairy Nasopharyngeal Polyp of Congenital Origin.

LEVINGER (*Münch. med. Wochenschr.*, No. 19, 1908) describes a case occurring in a 6½ months old female child. The tumor was pedunculated, 6 cm. long and 1½ cm. wide. It was covered with a delicate hairy epidermis. Through its middle was found a plate of cartilage. The growth was easily removed with the cold snare. Twenty-five case are known in the literature. *Horn.*

A Contribution to the Therapy of Angina.

BERLINER (*Deutsch med. Wochenschr.*, No. 13, 1908), uses a protargol salve combined with menthol, a small portion of which is introduced into the nasal aperture several times a day and appears to favorably affect the course of catarrhal and parenchymatous form. No cases are quoted. *Horn.*

The Local Treatment of Acute Inflammations of the Throat from the Standpoint of Pathology.

J. L. GOODALE, Boston (*The Boston Med. and Surg. Jour.*, June 25, 1908), has made a number of careful clinical examinations, both before, during and after treatment of acute inflammations of the throat, to see what the effect of treatment would be, local antiseptics and silver salts being used. As a result of his observation, he comes to the following conclusions:

1. "In a beginning tonsillitis antiseptic applications may perhaps be used with benefit and their effect, if any, will be to abort the local infection. If the disease is not checked at the outset by the sterilization of the parts, but if it proceeds to the formation of white spots in the crypts with systemic involvement, further application of antiseptics may not only be useless but harmful.

"It would appear possible that antiseptics may retard convalescence in two ways: First, by diminishing the number of bacteria in the crypts which are generating toxin and consequently prolonging the period required for the formation of the requisite amount of antibodies; second, by their destructive action upon the tissue cells and phagocytic leucocytes of the host.

2. "Forcible application of antiseptics may be followed by increased fever and cervical adenitis, indicating heightened

absorption of toxin into the system. The phenomenon may be compared to the depression which follows the injection of too large a dose of vaccine in cases undergoing opsonic treatment.

3. "In certain cases where acute tonsillitis appears to be aborted by local antiseptics, inflammatory manifestations may follow after a day or two in the neighboring regions and last for a number of days or several weeks. Here the possibility is suggested that the checking of the tonsillitis checked also the establishment of immunity, and that for its final accomplishment a longer period of growth of the organism upon the membranes of the nose, larynx, trachea or bronchi was necessary."

"If the conclusions to which I have come prove, on fuller investigation, definitely established, we shall have to modify our time-honored treatment of these affections. If I may venture to prophesy, our procedure will be approximately as follows: Active early local treatment as at present with guaiacum, silver preparations, etc., the avoidance of antiseptics when once the disease is definitely under way and, above all, complete local and general rest. It may be said with truth that the most vigorous and active measure that we can adopt in acute infections of the throat is a consignment of the patient to his bed, with avoidance of meddlesome interference."

Richards.

V—MISCELLANEOUS.

The Connection Between Disturbances of the Upper Air Passages and Diseases of the Genito-Urinary System.

SENDZIAK (*Mediz. Klinik,* No. 9, 1908) points out that diseases of the kidneys more often show themselves in the mucous membranes of the mouth and pharynx, than in the nose. We must be particularly careful, when a local cause is lacking, to regard with suspicion pallor of the membranes and unexplained hemorrhages, and examine the urine at once. In females, nasal disturbances of a genital origin are far more common; bleedings, hyperemia, secretion are commonly seen.

Horn.

Sodium Perborate.

HEYDENRICH (*Deutsch. med. Wochenschr.,* No. 3, 1908) recommends its use in ear discharges and as a post-operative powder in nasal work. He claims for it a certain hemostatic action. *Horn.*

HARTMANN (*Deutsch. med. Wochenschr.*, No. 7, 1908) warmly recommends its use as a dusting powder in internal diseases of the larynx. *Horn.*

A Pin in the Left Bronchus.

v. SCHROETTER (*Deut. med. Wochenschr.*, No. 15, 1908) gives an interesting case of a 12-year-old girl who aspirated a shawl pin 74 mm. long with a glass head 8 mm. in diameter. The pin was embedded for twenty-one days and removed by a direct bronchoscopy, through a tube 21 cm. long and 8.5 mm in diameter, at the first trial. No reaction; complete recovery. *Horn.*

A Universal Demonstration Lamp for Laryngoscopy, Tracheaoscopy and Esophagoscopy.

HORN, Bonn (*Deut. med. Wochenschr.*, No. 17, 1908). The handle was designed for use with the Kirstein electrical headlight. By a simple vertically placed joint, the lamp can be swung out the line of the tube and the mandrin removed, a cocain application made, or the coughing of pus against the mirror prevented. It was designed especially to replace the Casper electroscope, which because it employs a prism to cast the light at right angles down the tube occupies half the field of view and when once in position can not be removed for manipulative procedure, thus making it impossible for demonstrative work. *Horn.*

Bronchoscopy and Foreign Bodies.

VON EICKEN (*Deutsch. med. Wochenschr.*, No. 17, 1908) gives a short but very interesting review of the work of the Killian clinic for ten years, in the removal of foreign bodies from the bronchi. In fifteen cases, the foreign body was found twelve times. Twice it was impossible to certify the diagnosis and once the severe wound in the bronchus could be only explained as a trauma resulting from a foreign body. Seven cases were operated on with local anesthetics and eight in general narcosis. Of these eight cases four were operated on in America where the use of a general anesthetic is common and the other four were all children. The great advances which have been made are due largely to Killian's assistant, Brünnings, whose telescope tubes and electrical operating lamp leave little to be desired. One fatal case only mars the statistics; a baby who had aspirated a large bean and was brought into the clinic three days after the accident with a severe pneumonia. *Horn.*

The Operative Procedure in Deep Lying Abscesses of the Tongue.

BRUNK (*Deutsch. med. Wochenschr.*, No. 23, 1908), recites two cases and gives literature. He claims that the opening of the abscess from the mouth is uncertain and dangerous. Recommends the external route through the musculus hypoglossus. *Horn.*

Lactic Acid Bacilli Cultures in Nose, Throat and Ear Work.

H. HOLBROOK CURTIS (*New York Med. Record,* July 11, 1908) has been experimenting with culture of Massol's bacillus, introduced directly into the nasal cavities in cases of empyema of the accessory cavities, and in atrophic rhinitis. A cubic centimeter of the culture broth is sprayed under pressure so as to reach the ethmoid cell orifices, the theory being that the bacilli will find their way into the ethmoid cells, and accessory cavities, and by their secretion destroy the pyogenic bacteria which are causing the disease. Five cases of sinus and ethmoid trouble, and two of atrophic rhinitis are cited, in all of which rapid improvement took place. The announcement is preliminary only. *Richards.*

ANNALS

OF

OTOLOGY, RHINOLOGY

AND

LARYNGOLOGY.

VOL. XVII. DECEMBER, 1908. No. 4.

XXXIV.

THE IMPORTANCE OF THE UPPER RESPIRATORY TRACT IN THE ETIOLOGY OF CRYPTOGENETIC INFECTIONS, ESPECIALLY IN RELATION TO PLEURITIS.

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The work which is outlined in this paper was undertaken with the purpose of determining whether there could be any direct relation between lesions in the upper respiratory tract and plural infections. During the last few years several articles have appeared, pro and con, as to the possibility of a direct lymphatic connection between the throat and the bronchial glands as well as the pleural apices, but a great deal more thought and work have been directed toward the importance of the throat as being the starting-point for general hematogenous infection. These two questions are, however, closely associated, for if the invading germ has penetrated along the lymph-paths as far as the pleura, it would seem that occasionally an entrance into the blood might very easily be accomplished. And, on the other hand, if the pleura should be, at the time of the infection, the place of least resistance, bacteria which have entered the blood may lodge and grow at this spot. Yet, while there is more or less connection between the hematogenous and lymphatic routes, there may be a radical difference in their reception which is of great importance. Boeri has shown that while toxins pass through blood-vessel walls very easily, it is almost impossible for bacteria to do so, although they readily penetrate along the lymph-channels. If the infection is hematogenous in the start, it must be simply a septicemia due to the absorbing of toxins from an infecting focus, in which case only the toxins leave the original local process and not bacteria. If the lymph-channels are the route of invasion, then it is very possible for micro-organisms themselves to be carried from their original seat, and after successfully overcoming the various lymph-glands in their way, finally give rise to a true bacteremia. Therefore, in regard to the influence of lesions of the upper respiratory tract on pleural conditions we must recognize that there are two routes of attack from the throat.

For an infection to start there must be some portion of the throat which may act as a gate of entrance, some place of insufficient resistance which may either become an infecting nidus or act as an entrance. As lymphatic tissue is the tissue par excellence for the propagation of infections, it is natural that one should suspect the tonsils of showing lessened resistance.

By the tonsils I do not mean the faucial tonsils alone, but also the pharyngeal and lingual tonsils, the lymphoid tissue of the lateral folds of the pharynx, and the lymph-follicles of the posterior pharyngeal wall. However, as the faucial tonsils would generally represent by far the greater amount of lymphoid tissue in the throat, these structures are much more open to investigation, and hence the greater part of the work that will be outlined in this paper pertains especially to them.

The opposition to the entrance of bacteria into the body from any surface depends upon the vital resistance of the tissues, and also upon the mechanical barrier presented by the lining membrane. The epithelium lining the upper respiratory tract is either a stratified squamous variety, as exemplified in the mouth, or a stratified ciliated columnar type, as found in the nose. The surface of the faucial tonsils is covered by stratified squamous cells. The structure of a tonsil resembles somewhat that of a lymph-gland, differing, however, in that it possesses no afferent lymph-vessels and no periglandular sinuses, and does possess those peculiar structures, the crypts or lacunae.

The crypt of the tonsil is its peculiar and most characteristic structure. It consists of an invagination of the epithelium from the surface of the tonsil, which has undergone a very interesting anatomical change. In the first place, the subepithelial connective tissue which exists in a marked degree beneath the surface epithelium, disappears as soon as the epithelium starts to form the crypts. This permits the epithelium to come in direct contact with the lymphatic structure of the tonsil, and very frequenly it is impossible to distinguish a dividing line between the epithelium of the crypt and the interfollicular tissue. The epithelium of the crypt, unlike its progenitor which covers the surface of the tonsil, does not form a compact, unbroken barrier of protection. For the greater part of its extent it presents an intact line, only one or two or probably three cells in thickness, and there are occasional spaces between these cells. Toward the parenchyma the epithelial cells show a peculiar condition. They are separated from each other by interposed cells varying in type from slightly changed epithelial cells to well-formed lymphocytes. The epithelial cells may also extend from the crypt into the tonsillar substance, suggesting the ramifications of a malignant epithelioma. The smaller terminal invaginations of the cryptal epithelium are usually solid sprouts, sometimes, however, with central keratosed cores, the exfoliation of which causes an extension of the lumen of the crypt.

In the crypt, then, we have a loose, disintegrated epithelium which, as far as any mechanical barrier is concerned presents an open door to microbic infection, and it has been shown that minute inert foreign bodies readily pass between the cells. Further, in the faucial tonsils especially, the gross arrangements of the crypts is such that frequently they form pockets and recesses capable of retaining large numbers of bacteria which find plenty of nourishment in the desquamating epithelium..

Now, it would seem that unless the vital resistance is considerably greater in the tonsil than in the surrounding structures, the tonsillar tissue ought to be the vulnerable spot in the throat. Subject as the tonsil parenchyma is to microbic influences by absorption of toxins, and also, it may be, of bacteria through the cryptal walls, it was thought possible that there might be developed in the tonsil certain antibodies which would make this structure especially resistant to germ invasion. If it could be demonstrated that the tonsils held in store great quantities of antitoxin and bacteriolytic substances, then one might possibly be justified in believing that the tissue of which they are composed is especially resistant to bacterial invasion.

The following experiments were kindly carried out for me by Dr. M. P. Ravenel. The chief difficulty which seemed to stand in the way of this research was to obtain the soluble portions of the tonsil in a sterile condition, without destroying the immune bodies if such existed, but this was overcome by the use of the Pasteur filter.

About 13 g. of hypertrophied faucial and pharyngeal tonsils, removed from several children, were ground to a pulp in a "broyeur" with about 50 c.c. of sterile water. The mixture was then passed through a Pasteur filter. A part of the filtrate, as a control test, was incubated for one week, and at the end of that time was still clear and sterile.

The rest of the filtrate was treated as follows:

One cubic centimeter of filtrate was added to 5 c.c. of bouillon and inoculated with typhoid bacilli.

Two cubic centimeters of filtrate were added to 5 c.c. of bouillon and inoculated with typhoid bacilli.

After incubation all of these tests showed the same degree of turbidity in the same length of time.

In these experiments it was supposed there was sufficient complement present in the tonsils to cause lysis of the typhoid bacilli, provided the specific immune body was present in sufficient quantity to unite it with them. This was, on the face of it, an unwarranted supposition, and again the known frailty of complements in general was a very poor guarantee for its safe passage through all the manipulation and time requisite to sterilize the tonsillar substance.

Therefore a second series of tests was made. The sterile tonsillar filtrate was obtained as before, the amount of tonsillar substance being the faucial and pharyngeal tonsils from three children in whom hypertrophy of these structures had made operation necessary.

It might very well have happened that the children from whom the tonsils were removed had never been brought in contact with the typhoid bacillus, and consequently had no specific immunity to this disease, so in this second series a more common micro-organism was employed.

Ten tubes of bouillon were inoculated with the staphylococcus aureus. To eight of these tubes the tonsillar filtrate was added, and to four of these eight fresh serum was added. All ten tubes were then incubated for twenty-four hours, and all showed the same degree of turbidity, that is, equal bacterial growth.

This failure to find immune bodies in the tonsil may be due to a lack of sensitiveness in the technic; or to the simple fact that the reasoning concerning this subject has been false; or it may be that soluble substances are so easily removed by both the blood-vessels and lymph-channels that immune bodies as well as toxins and antitoxins cannot accumulate in the tonsils. To my reasoning this last explanation would seem the most likely. The blood-supply of the tonsil is fairly rich and the efferent lymph-stream is a decided entity, so that a soluble substance could probably not remain long in the tonsillar structure. The rapid and marked systemic intoxication seen in cases of simple tonsillitis shows that some toxins, at least, are readily absorbed from the tonsil into the general circulation, and it is more than probable that the first general symptoms are due to toxic absorption through the blood-vessels. The slowness of the lymphatic method of infection is shown by the fact that enlargement and tenderness of the tonsillar lymph-gland at the angle of the jaw is not always present in simple tonsillitis, and is almost never present at the very beginning of the attack.

It seems, therefore, scarcely possible for a great quantity of immune bodies to accumulate in the tonsil, and it is much more probable that the larger amount of such material enters the general circulation. This failure to find any considerable amount of immune bodies in the tonsillar tissue, as has been indicated, leaves us still uncertain as to the degree of vital resistance of the tonsillar structure.

Of later years there has been a great deal of work done in regard to the susceptibility of the tonsil to tuberculous infection, but as I have already gone over the literature of this subject rather thoroughly as late as 1904, I will simply mention some of the deductions and give the bibliography which bears on the subject of this paper.

In pulmonary tuberculosis the expectoration is coughed up so that it comes in nearly equal contact with all parts of the respiratory tract except the nose and nasopharynx, and thus a fairly even dose of tubercle bacilli is spread over the mouth, pharynx, and larynx. In one hundred and thirty-six cases of pulmonary tuberculosis reported by seven different observers, the tonsils were involved ninety-four times. That is, in 69 per cent of pulmonary tuberculosis the tonsils were diseased. In 1904 I reported nine cases of pulmonary tuberculosis in which the tonsils were examined at autopsy, and in every one I found histologic changes sufficient to justify a diagnosis of tuberculosis. This evidence is pretty strong, showing that, at least as far as tubercle bacilli are concerned, the tonsil is much more easily invaded than any other part of the upper respiratory tract. This argues against the theory that the tonsillar tissue is especially resistant to bacteria. The question, however, is so important that it was thought worth while to examine a sufficient number of cases to positively determine the tonsillar vulnerability to the tubercle bacillus at least; so during the past year I made a careful post-mortem study of the tonsils of thirty-seven cases, who clinically were supposed to have died from pulmonary tuberculosis.

These cases are reported in some detail as to the histological condition of the tonsils, with a brief synopsis of the general tuberculous involvement. The numbers attached are the regular numbers at the Phipps Institute.

TUBERCULOSIS OF THE LUNGS WITH TUBERCULOSIS OF THE TONSILS.

Case No. 903. Left pleura showed adhesions everywhere. Left lung, upper lobe showed a large cavity and infiltration, lower lobe was partially consolidated. Right pleura showed dense adhesions everywhere. Right lung, upper lobe showed numerous cavities with excess of fibrous tissue, and the middle and lower lobes a few isolated tubercles. The liver, spleen, and kidneys showed tuberculous lesions.

Tonsils were very small and portions of the cryptal epithelium infiltrated. The germinating follicles were few and small. There was marked fibrosis. A few discrete but distinct tubercles were found, showing caseation and typical giant cells.

Case No. 2632. Left pleura was thickened, with adhesions at apex. Left lung, upper lobe showed one large cavity with several small ones, lower lobe was compressed and infiltrated. Right pleura was thickened, with adhesions everywhere. Right lung, upper lobe was pneumonic, with numerous tubercles, middle and lower lobes were partially infiltrated. The liver, intestines, kidneys, and lymph-nodes showed tuberculous involvement.

Tonsils were small with only slight infiltration of the cryptal epithelium and very few germinating follicles. There were discrete tubercles with giant cells.

Case No. 2727. Left pleura showed adhesions. Left lung, upper and lower lobes showed cavity formations and coalesced tubercles. Right pleura showed adhesions at apex. Right lung, upper lobe showed one large cavity, middle and lower lobes were infiltrated, with coalesced and miliary tubercles. Spleen was tuberculous.

Tonsils were moderate in size, congested, and the cryptal epithelium was infiltrated. There was an excess of lymphoid tissue which was infiltrated with polymorphonuclear leucocytes. There were diffuse tubercles with caseation and giant cells.

Case No. 2980. Left pleura showed adhesions everywhere. Left lung, upper lobe showed cavities and consolidation, lower lobe was congested and infiltrated with both coalesced and miliary tubercles. Right pleura showed adhesions everywhere. Right lung, upper lobe showed a cavity and was infiltrated, middle lobe was edematous, lower lobe showed a few coalesced tubercles. Liver, kidney, and spleen were tuberculous.

Tonsils were large and showed considerable lymphoid tissue. There were numerous diffuse and discrete tubercles showing necrosis and giant cells.

Case No. 3052. Left pleura was thickened, with adhesions everywhere. Left lung, upper lobe showed a cavity and pneumonic consolidation, lower lobe showed numerous tubercles. Right pleura showed a few adhesions at apex. Right lung, upper, middle, and lower lobes contained a few tubercles. Larynx was tuberculous.

Tonsils were moderate in size, with numerous follicles. The cryptal epithelium was infiltrated and there was a slight fibrosis. There were a few tubercles with necrosis and giant cells.

Case No. 3182. Left pleura showed adhesions. Left lung, upper and lower lobes showed numerous cavities and were consolidated. Right pleura showed a few adhesions. Right lung, upper, middle, and lower lobes all showed cavity formation, with more or less pneumonic consolidation. Intestines and kidneys were tuberculous.

Tonsils were very small. The cryptal epithelium was intact and infiltrated. There were no follicles and very little lymphoid tissue. There was marked diffuse tuberculosis with caseation and numerous giant cells.

Case No. 3253. Left pleura showed adhesions over the upper lobe. Left lung, upper lobe showed a small cavity, areas of consolidation, and some excess of fibrous tissue. Right pleura showed adhesions over the upper and lower lobes. Right lung, upper, middle, and lower lobes showed numerous cavities, with consolidation and caseation.

Tonsils were small, had no follicles, and very little lymphoid tissue. There was considerable fibrosis. The epithelium of the crypts was not infiltrated, but the crypts were filled with masses of bacteria, and portions of the epithelium were ulcerated. There were several cartilaginous nodules in the connective tissue. After cutting numerous sections a few tubercles were found showing typical giant cells and caseation.

Case No. 3393. Left pleura showed adhesions throughout. Left lung, upper and middle lobes showed cavity formation and caseous consolidation. Right pleura showed adhesions at apex. Right lung, upper and middle lobes showed small cavities and irregular consolidation. Larynx, intestines and kidneys were tuberculous.

Tonsils were small. The cryptal epithelium was for the most part not infiltrated. There was very little lymphoid tissue and a very few small follicles. There was marked fibrosis. Numerous discrete tubercles were found with giant cells but no necrosis.

Case No. 3405. Left pleura was normal. Left lung, upper lobe showed small tubercles, lower lobe showed caseous pneumonia. Right pleura showed adhesions at apex. Right lung, upper lobe showed one large cavity and infiltration, middle and lower lobes were partially consolidated. Liver and spleen were tuberculous.

Tonsils were very small. The epithelium of the crypts was considerably infiltrated and follicles were moderately numerous. There was slight fibrosis. Numerous small discrete tubercles were seen and a great many giant cells.

Case No. 3441. Left pleura showed adhesions at apex. Left lung, upper lobe showed several cavities and was infiltrated. Right pleura showed adhesions around the upper lobe. Right lung, upper and lower lobes showed cavity formation, middle lobe showed a few isolated tubercles. Kidneys were tuberculous.

Tonsils were very small. The epithelium for the most part was intact, but was ulcerated in one or two places. There was very little lymphatic tissue and the follicles were few and small and there was marked fibrosis. Several tubercles were found, some with caseation and giant cells.

Case No. 3526. Left pleura showed adhesions everywhere. Left lung, upper lobe showed a cavity and was partially infiltrated. Right pleura showed adhesions everywhere. Right lung, upper, middle, and lower lobes showed cavity formation. Trachea, intestines, and kidneys were tuberculous.

Tonsils were small. There were a few follicles and the epithelium of the crypts was mostly intact. There was considerable fibrosis. There were several discrete tubercles with giant cells.

Case No. 3548. Left pleura showed adhesions about the upper lobe. Left lung, upper and lower lobes showed cavity formation with some caseous pneumonia. Right pleura showed adhesions throughout. Right lung, middle and upper lobes showed cavity formation with some consolidation, lower lobe contained a few coalesced tubercles. Larynx and intestines were tuberculous.

Tonsils were small. The epithelium was generally intact, but necrotic in a few places. The follicles were few and small. There were numerous tubercles with giant cells, but very little caseation.

Case No. 3559. Left pleura showed adhesions over upper lobe. Left lung, upper lobe showed cavity formation, lower lobe was partly consolidated. Right pleura showed extensive adhesions. Right lung, upper, middle and lower lobes showed rather extensive cavity formation. Larynx, trachea, intestines, and kidneys were tuberculous.

Tonsils were small, containing few follicles, and the cryptal epithelium was intact. There was considerable fibrosis. There were numerous diffuse tubercles with giant cells.

Case No. 3633. Left pleura was normal. Left lung, upper and middle lobes showed numerous miliary tubercles. Right pleura showed adhesions at apex and on diaphragm. Right lung, upper lobe showed several cavities, middle and lower lobes showed miliary tuberculous infiltration. Larynx, intestines, and spleen were tuberculous.

Tonsils were small and contained only a few small follicles. The cryptal epithelium was slightly infiltrated, and that only in a few places. There was considerable fibrosis and there were some cartilaginous nodules in the connective tissue. A few scattered tubercles with giant cells were found.

Case No. 3643. Left pleura showed adhesions at apex. Left lung, upper lobe showed cavity formation, lower lobe contained a very few tubercles. Right pleura showed adhesions at apex. Right lung, upper and lower lobes contained cavities, middle lobe showed a few isolated tubercles. Liver and intestines were tuberculous.

Tonsils were somewhat enlarged and there was considerable amount of lymphatic tissue, but very few follicles. The epithelium was infiltrated. There was some congestion with very slight fibrosis. Several rather diffuse tubercles were found, showing caseation and nuclear fragmentation, but there were no typical giant cells.

Case No. 3677. Left pleura showed adhesions at apex. Left lung, upper and lower lobes contained cavities. Right pleura showed adhesions throughout. Right lung, upper, middle, and lower lobes all showed cavity formation.

Tonsils were moderate in size. The cryptal epithelium was intact and there were very few and small follicles. There was slight fibrosis. There was a diffuse tuberculosis, with a few giant cells, considerable nuclear fragmentation, but little necrosis.

Case No. 3697. Left pleura showed adhesions throughout. Left lung, upper lobe contained a large cavity, lower lobe showed numerous coalesced tubercles. Right pleura showed extensive adhesions. Right lung, upper lobe contained a large cavity, middle lobe contained numerous coalesced tubercles, lower lobe showed very little consolidation. Larynx, intestines, liver and kidneys were tuberculous.

Tonsils were small. The follicles were very few and small. There was considerable fibrosis, though the epithelium was for the greater part infiltrated. Numerous tubercles with giant cells were found and some caseation.

Case No. 3749. Clinical diagnosis was pulmonary tuberculosis with pneumothorax. Unfortunately there were no postmortem notes as to the condition of the lungs.

Tonsils were moderate in size, with fair amount of lymphoid tissue, but no follicles. The epithelium was for the greater part infiltrated and there was a slight fibrosis. Numerous discrete tubercles were found, with numerous and very large giant cells.

Case No. 3831. Left pleura showed adhesions at apex. Left lung, upper and lower lobes showed cavity formation with caseation. Right pleura was normal. Right lung, upper, middle and lower lobes showed a few tubercles.

Tonsils were small. There was a great deal of fibrous tissue. The epithelium was infiltrated only in places. There was very little lymphoid tissue and practically no follicles. There were fairly numerous discrete tubercles, with giant cells, but no necrosis. Some of the tubercles were evidently being replaced by fibrous tissue.

Case No. 3880. Left pleura showed extensive adhesions. Left lung, upper lobe contained a cavity and some infiltration, lower lobe was infiltrated. Right pleura showed adhesions throughout. Right lung, upper lobe contained a cavity and considerable fibrous tissue, middle and lower lobes contained tubercles, some of which were healed.

Tonsils were medium in size. The follicles were fairly numerous and about ordinary size. The epithelium was infiltrated. There was very little fibrosis. A few tubercles, with giant cells and necrosis, were found.

Case No. 3887. Left pleura showed few adhesions. Left lung, upper and lower lobes contained a few scattered tubercles. Right pleura showed adhesions at apex. Right lung, upper lobe contained a large cavity with some infiltration, middle and lower lobes presented a few isolated tubercles.

Tonsils were small. The epithelium was infiltrated in places; there was marked fibrosis, but fairly normal and fairly large follicles; also some congestion. A very few but distinct tubercles were found with giant cells and caseation. Some of the sections showed no tuberculosis.

Case No. 3907. Left pleura showed adhesions at apex. Left lung, upper lobe contained several cavities, lower lobe was partially infiltrated. Right pleura showed adhesions at apex. Right lung, upper, middle, and lower lobes all contained cavities.

Tonsils were small, the follicles were few and small. The epithelium was slightly infiltrated and there was considerable fibrosis with very little lymphoid tissue. A few tubercles with giant cells were found.

Case No. 3918. Left pleura showed extensive adhesions. Left lung, upper and lower lobes showed considerable infiltration. Right pleura was not reported on. Right lung, upper, middle, and lower lobes were all more or less infiltrated.

Tonsils were moderately enlarged. The epithelium was infiltrated in only a few places. The follicles were fairly numerous and quite large. There was slight fibrosis and considerable congestion. A very few tubercles, with giant cells, were found.

Case No. 3941. Left pleura showed adhesions at apex. Left lung, upper lobe contained numerous cavities, lower lobe was partially consolidated. Right pleura showed adhesions everywhere. Right lung, upper, middle, and lower lobes all showed cavity formation with infiltration.

Tonsils were rather large, but with very few follicles. The epithelium of the crypts was somewhat infiltrated and there was slight fibrosis. There was marked tuberculosis, with numerous tubercles showing giant cells and caseation.

Case No. 3953. Left pleura showed adhesions everywhere. Left lung, upper and lower lobes both showed cavity formation. Right pleura showed adhesions everywhere. Right lung, upper, middle, and lower lobes all showed cavity formation. Larynx and intestines were tuberculous.

Tonsils were slightly enlarged. The epithelium of the crypts was fairly intact and there was marked fibrosis and few follicles. There was very marked tuberculous infiltration, consisting of both discrete and confluent tubercles. There was some caseation, but the tubercles seemed to be undergoing fibrosis rather than softening. There were numerous giant cells.

Case No. 4010. Left pleura showed adhesions everywhere. Left lung, upper lobe showed numerous cavities with some excess of fibrous tissue. Right pleura showed adhesions at apex. Right lung, upper lobe showed cavities with excess of fibrous tissue, middle lobe showed a few tubercles, lower lobe showed one cavity with a few tubercles. Larynx, intestines, and kidneys were tuberculous.

Tonsils were rather large, with few follicles. The cryptal epithelium was considerably infiltrated and there was marked fibrosis. There were large numbers of discrete tubercles, with numerous giant cells, but very little necrosis.

Case No. 4021. Left pleura showed adhesions in the upper part. Left lung, upper lobe showed cavities, lower lobe showed numerous coalesced tubercles. Right pleura showed adhesions at the apex. Right lung, upper, middle, and lower lobes showed cavities and caseous tubercles. Kidneys were tuberculous.

Tonsils were very small, with a very small amount of lymphoid tissue. The epithelium was intact, and there was marked fibrosis. There were only a few doubtful tubercles.

Case No. 4040. Left pleura showed adhesions at apex. Left lung, upper and lower lobes were almost completely consolidated. Right pleura showed extensive adhesions. Right lung, upper and lower lobes showed cavity formation, with infiltration, middle lobe was infiltrated. Larynx, liver, intestines, and kidneys were tuberculous.

Tonsils were rather large, but there was only a small amount of lymphoid tissue. The cryptal epithelium, though generally intact, was ulcerated in a few places. There was very marked fibrosis and the follicles were few and small. There were numerous tubercles, both discrete and confluent, with giant cells and considerable caseation.

Case No. 4141. Left pleura showed old adhesions. Left lung, upper lobe showed a healed lesion, lower lobe showed bronchopneumonia. Right pleura showed old adhesions. Right lung, upper lobe showed a large cavity, with consolidation, which was not typical, however, of tuberculosis; middle and lower lobes showed bronchopneumonia. Kidneys and spleen showed doubtful tuberculosis.

Tonsils were small. Cryptal epithelium was slightly infiltrated. Follicles were small, but fairly numerous, and there was no fibrosis. There were a few very doubtful tubercles found.

Case No. 4155. Left pleura showed adhesions everywhere. Left lung, upper and lower lobes showed cavity formation and caseous infiltration. Right pleura showed adhesions at apex. Right lung, upper and middle lobes showed cavity formation with fairly numerous tubercles. Larynx, liver, and intestines were tuberculous.

Tonsils were very small and had very few and small follicles. There was marked fibrosis and the epithelium was generally intact. There were a few tubercles with giant cells, but no necrosis.

Case No. 4281. Left pleura showed some adhesions along the axillary line. Left lung, upper lobe showed a cavity and was infiltrated, lower lobe was partially consolidated. Right pleura showed adhesions in axillary line. Right lung, upper, middle, and lower lobes all showed tuberculous infiltration. Larynx, trachea, liver, intestines, kidneys and spleen were tuberculous.

Tonsils were small and the follicles were very few and small. The epithelium was active in some places and there was considerable fibrosis. There were a few distinct discrete tubercles with giant cells, but no fibrosis.

TUBERCULOSIS OF THE LUNGS WITHOUT TUBERCULOSIS OF THE TONSILS.

Case No. 3896. Left pleura showed a few adhesions. Left lung was practically normal except for a small healed lesion and a few miliary tubercles. Right pleura showed adhesions at apex. Right lung, upper lobe showed cavities and some consolidation, middle and lower lobes were partly consolidated.

Tonsils were somewhat enlarged. There were numerous follicles. The epithelium of the crypts was infiltrated. One of the crypts contained a calculus. There was slight fibrosis and some cartilage in the connective tissue; also some congestion. No tuberculosis was found.

Case No. 4023. Left pleura showed adhesions everywhere. Left lung, upper lobe showed a cavity, with excess of fibrous tissue, lower lobe showed a few isolated and coalesced tubercles. Right pleura showed adhesions everywhere. Right lung, upper, middle, and lower lobes all showed cavity formation. Liver and large intestine showed tuberculosis.

Tonsils were extremely small and showed almost no lymphoid tissue or follicles. The epithelium was almost everywhere intact. No tuberculous changes were found.

Case No. 4140. Left pleura showed extensive adhesions. Left lung, upper and lower lobes were infiltrated. Right pleura showed extensive adhesions. Right lung, upper, middle, and lower lobes showed tuberculous infiltration. Intestines were tuberculous and showed a perforating ulcer which by causing peritonitis was the evident cause of death. Kidney and spleen were also tuberculous.

Tonsils were moderate in size. Crypts were generally intact and there were only a few small follicles, but marked fibrosis. No tuberculosis was discovered.

NON-TUBERCULOUS CASES.

Case No. 3537. Left pleura showed adhesions at apex, but the case was not proved at autopsy to be tuberculous. There were scattered areas of pneumonia in each lung, and numerous small foci, containing a thick, creamy, pus-like substance, and a few other areas which looked suspiciously like tubercles.

Tonsils were large, containing numerous and fairly large follicles. The epithelium was greatly infiltrated, and there was a slight fibrosis. No tuberculous changes were discovered.

Case No. 3699. There was a pleuritic effusion on the left side, with hypostatic pneumonia in the lower lobe. The right lung was nearly normal. Patient died of valvular heart disease.

Tonsils were large, with infiltrated epithelium, a slight fibrosis, marked congestion, and small but fairly numerous follicles. No tuberculous changes could be found.

Case No. 4116. Left pleura was normal. Left lung, upper and lower lobes showed no tuberculosis. Right pleura showed adhesions throughout, and there was a purulent pleurisy. Right lung was collapsed, but showed no tuberculosis. The spleen showed areas of possibly healed tubercles.

Tonsils were large. There was considerable lymphoid tissue and numerous follicles. The epithelium was greatly infiltrated and there was a slight fibrosis. No tuberculosis could be found.

In the thirty-seven cases just outlined, three were found on post-mortem examination to be non-tuberculous. The tonsils of each of these non-tuberculous cases failed to show histological changes at all characteristic of tuberculosis. In the rest, that is, in thirty-four cases, the tonsils of twenty-nine showed absolutely typical lesions of tuberculosis, and two showed lesions which were probably tuberculous, though no typical giant cells were found. In the remaining three of the thirty-four tuberculous cases no change at all characteristic of tuberculosis could be found in the tonsils. In these three cases six to ten slides were examined; all, however, were cut from the same paraffin block, so that by far the greater part of the tonsils was not examined, and it is possible that some portion of the tonsils which was not examined might have been tuberculous.

So far it seems practically proved that for the tuberculosis germ, at least, the tonsil is many more times susceptible than other portions of the throat. Further, I believe that it is a justifiable deduction to say that if one organism passes readily into the tonsillar tisue it is probable that other organisms may do the same thing, provided their pathogenic power is sufficiently great to overcome the tissue resistance. The findings in acute tonsillitis would tend to bear this out. In this disease the number of micro-organisms which invade the parenchyma of the tonsil is so large that they can easily be found with the microscope, and the varieties of bacteria which are capable of producing tonsillitis are very great, including practically all of the pyogenic micro-organisms.

The crypts of normal tonsils are frequently filled with a large number of micro-organisms, but they are seldom found in the parenchyma. The only method, however, of direct investigation—that of microscopic examination—is most unsatisfactory on account of its limitations, in that if only a few micro-organisms are present they may readily escape detection. Inoculation or culture tests are of absolutely no value, as the cryptal ramifications are so numerous and so close together that it would be impossible to obtain any considerable portion of the tonsil without a crypt, and hence contamination.

E. Lexer claims by experimentation to have succeeded in getting streptococci, which had been cultivated so as to have a special virulence toward the rabbit, to enter the crypts, pass through the tonsillar tissue, and to enter the blood within three and a half hours after inoculation. The organisms were found in the tonsillar substance an hour after inoculation.

Piera also demonstrated experimentally that micro-organisms could be absorbed through the epithelium of the crypts, but if they were not capable of resisting the vital action of the tissues they were destroyed practically as soon as they had entered the tonsillar parenchyma.

From the clinical standpoint there is strong evidence that various organisms not only penetrate into the tonsillar substance, but also pass from the tonsil to the general system. The variety of diseases which has been reported as resulting from tonsillar infection includes acute inflammation of nearly all organs. The literature on this subject is rather excessive, but it is well worth while to briefly review some of these articles, as they not only point out the possibility of systemic infection through the tonsils, but are also of value in that they give a more comprehensive view of the subject, representing a greater extent of thought.

I will take up the various diseases separately, giving under each a brief abstract of the individual papers.

RHEUMATISM.

Acute inflammatory rheumatism is now considered by the large majority of clinicians to be an infectious disease, and the possibility that in a certain number of cases the infection arises within the tonsil is being more and more recognized.

Suchannek believes that a considerable number of these cases are caused by tonsillitis.

Meitus says that articular rheumatism is caused by micro-organisms which enter the body through the tonsils.

Packard, after a careful and critical review of the literature on this subject, believes it is possible that there is an attenuation of micro-organisms invading the body through the adenoid tissue of the throat, so that they produce the phenomenon known as articular rheumatism rather than the ordinary septicemia or pyemia.

St. Clair Thompson says that the connection of angina and rheumatism, though undoubted in a certain number of cases, is not yet clearly established. The tonsils may be the port of entry of the rheumatic virus, and this even though the naked-eye appearance of the throat gives no indication of its being affected.

Jakobi admits the close connection between tonsillitis and rheumatism, and advises the removal of large tonsils.

Stengel reports one case, and Turner two cases, of acute rheumatism, within a few days after an attack of sore throat.

In two hundred and seventy cases of acute rheumatism reported by McCrae, only 3.7 per cent had tonsillitis previous to the attack.

Kuhn reports three cases of acute articular rheumatism, following tonsillitis.

Williams and Miller both concede to the tonsils a very important part in the production of acute rheumatism.

A critical editorial in the "Journal of the American Medical Association" says that the tonsils seem to be in a large number of cases the portal of entry of the infection in articular rheumatism.

Gurich reports three cases of rheumatism directly traceable to tonsillar infection, and claims that many cases of acute articular rheumatism are caused by "fossulare angina," and that the rheumatism gets well when the tonsillar disease has been properly treated.

Ingalls, after a careful study of one hundred of his own cases of tonsillitis, reverses his former view, and now believes that tonsillitis plays a very important part in the etiology of rheumatism.

Phillips, in two hundred and ten cases of acute articular rheumatism, found that tonsillitis developed during or before the illness in 18.01 per cent.

Epstein reports one case of rheumatism complicated with pleurisy, following an attack of tonsillitis.

Hammerschmidt reports seventeen cases of rheumatism, in nine of which tonsillitis was present for three to seventeen days before the rheumatic outbreak, and in the rest the throat was more or less inflamed during the attack.

Menzer says that the entrance gate for bacteria in acute articular rheumatism is in the large majority of cases, though not all, the tonsils.

Adler says that rheumatic attacks may follow a very mild tonsillitis.

Miller reports two cases of polyarthritic inflammation, in which he believes the infection was due to frequent attacks of tonsillitis.

Bruck believes that in all forms of rheumatism the tonsils form the gate of entrance par excellence for the infection.

Trousseau was probably the first to show the relation between tonsillitis and rheumatism.

Lagranere and Pipounier showed that acute articular rheumatism was accompanied by rheumatic angina, which occurred sometimes before, sometimes with, and sometimes after, the beginning of the rheumatic attack.

Caesar Boeck reports an epidemic of angina lacunaris, during the convalescence of which the patients regularly developed acute articular rheumatism.

Lasegue reports several cases showing the relationship between these two diseases.

Brown describes a regular epidemic of tonsillitis in school children with rheumatic complications.

Stephanides reports a case of repeated attacks of rheumatism cured by extirpation of the tonsils.

Whipham collected six hundred and fifty-five cases of acute articular rheumatism in which inflammation of the tonsils played a most important part.

Roos reports six cases of angina followed by articular rheumatism.

Peltesohn says that "angina lacunaris, acute articular rheumatism, muscular rheumatism, and certain diseases of the skin occrring with these, are etiologically diseases very close to each other."

Goodale says that in a certain percentage of cases the good operative results following the removal of tonsils seem to justify the theory that the faucial tonsils may constitute an infection atrium for rheumatic arthritis.

Chronic Rheumatism.—Gurich has claimed to be able to differentiate between chronic articular rheumatism due to tonsillar infection and that due to other causes by removing the tonsil. In the tonsillar infection cases the operation is followed by a reaction in the involved joints. Out of one hundred and two cases of chronic articular rheumatism which were thus diagnosed as due to tonsillar irritation, ninety-eight were permanently healed by the removal of the tonsils.

Goodale has recently shown a relation between certain cases of chronic arthritis and the presence of retained debris in the tonsillar tissue.

GENERAL INFECTION.

Otten reports two cases of general staphylococcic infection originating from the tonsils.

Woodcock reports one case of pyemia and phlebitis following tonsillitis with abscess, which was cured with antistreptococcic serum.

Stengel, White and Evans report one case in which the streptococcus was found in the blood and in the tonsils.

Tishutkin reports one case of fatal septicemia originating from a collection of pus in the right tonsil.

Edson describes one case of septicemia which he believes was probably caused by an infection of the tonsil.

Wainwright reports one case of fatal pyemia following tonsillitis.

Owen reports one case of septicemia following tonsillitis.

Jessen reports four cases of general infection following tonsillitis.

Buschke reports four cases of systemic infection following tonsillitis.

Adler reports two fatal cases of general septic infection, in which he believes the infecting germ originated from a mild tonsillitis.

Anderson believes very strongly that the tonsils are frequently portals of infection.

Jacobi, while saying that the modern custom of making the tonsil exclusively responsible for the admission into the system of microbes and toxins is unjustifiable, admits that such invasion may occasionally take place.

Trautmann says that it is possible that many of the infectious diseases originate through the tonsils.

Obtulowicz has seen a case of general infection following angina.

Buss has seen a case of tonsillar inflammation followed by general infection and extensive phlegmon of the skin of the elbow.

Babes autopsied a case of septic infection of all the organs due to a moderately severe tonsillitis; streptococci were found in large numbers.

Buhorst describes two cases of severe infection developing after an angina.

NEPHRITIS.

Morse reports five cases of acute nephritis following tonsillitis.

Hammerschmidt reports five cases of nephritis following tonsillitis.

Adler says that 75 per cent of all cases of tonsillitis are accompanied by a mild type of nephritis, which occasionally becomes chronic.

Thouvenet reports one case of nephritis subsequent to an atack of acute tonsillitis.

Baduce reports four cases of angina followed by nephritis, in which the diplococci of pneumonia were found in the blood until the case was cured.

Jeppe saw five cases of angina with nephritis.

Czajkowski reports seven cases of acute nephritis associated with angina lacunaris.

TENOSYNOVITIS.

Tracy reports two cases of acute tenosynovitis following acute tonsillitis.

ENDOCARDITIS.

Ross reports one case of streptococcic endocarditis following tonsillitis. Streptococci were found both in the blood and in the tonsils. He believes that in most cases of infectious diseases tonsillitis is a prominent symptom.

Charrin reports one fatal case of staphylococcic endocarditis following an attack of acute tonsillitis.

Mayer reports one case of endocarditis following an attack of tonsillitis.

Packard reports five cases of endocarditis in which he believes that the infection gained access to the blood through the tonsils.

BRONCHITIS.

Hammerschmidt reports six cases of bronchitis occurring in healthy men, following tonsillitis.

PNEUMONIA.

Hammerschmidt reports two cases of bronchopneumonia and two cases of lobar pneumonia following tonsillitis.

Adler reports one case of streptococcic pneumonia resulting from a streptococcic tonsillitis.

Wallace reports one case of pneumonia following a severe inflammation of the throat.

PLEURISY.

Heddaeus reports one case of pleurisy and pneumonia following a staphylococcic tonsillitis. He believes the pneumonia to be a coincidence, the pleurisy due to the staphylococcus.

Huguenin reports one case in which double-sided pleurisy developed after a severe angina due to the staphylococcus pyogenes aureus.

Gougenheim reports one fatal case of pleurisy and pericarditis following streptococcus angina.

APPENDICITIS.

Forchheimer reports one case of appendicitis following tonsillar infection.

Freeman (discussing Forchheimer's paper) reports one case of appendicitis following tonsillitis.

Mitchell reports one case, coming on a few days after the onset of tonsillitis, and later followed by nephritis and endocarditis.

JAUNDICE.

Forchheimer reports five cases of jaundice following tonsillitis; three to ten days elapsing between the onset of the two diseases.

PHLEBITIS.

Trumbull reports one case of fatal phlebitis following tonsillar infection.

Wagner reports two cases from which an apparently slight angina produced a suppurative thrombosis of the jugular vein, followed by pyemia. Streptococci were found in the tonsils and vein.

Hager and Sahli report cases similar to those of Wagner.

MENINGITIS.

Hammerschmidt reports one case of meningitis due to Fraenkel's diplococcus, which organism was also found in the tonsil.

Perigord reports one case of fatal meningitis following abscess in the left tonsil.

De Guelder reports one case of streptococcic angina complicated with meningitis.

PAROTITIS.

Hammerschmidt reports twenty-five cases of parotitis, and all except one were accompanied sooner or later by tonsillitis; three before, fifteen at the same time, only six after the parotitis had set in.

SKIN LESIONS.

Le Gender reports four cases of erythema following tonsillitis.

Leven reports one case of scleroderma following tonsillitis.

Legendu and Claisse report one case of erythema developing during an attack of streptococcic tonsillitis.

Bruck reports four cases of rheumatica purpura following tonsillitis.

EYE LESIONS.

Vergely reports one case of phlegmon of the orbit, directly following acute tonsillitis.

While the above synopsis of literature concerning tonsillar infection is not a complete list of all that has been written on this subject, it is sufficiently extensive to show that the idea of systemic infection arising through the tonsils is a very general one and not limited to any individual or to any country. Further, a perusal of these articles has given me the distinct impression that the authors of these various papers have been impressed with the importance of the tonsil as a gateway for infection.

The importance of this literature to the thought which I have attempted to develop is that it unquestionably shows that if the micro-organism has access to the tonsil parenchyma, it is possible that it may pass from there into the blood. As has been stated by Boeri, it is the exception, if indeed it ever happens, that bacteria can pass through a normal blood-vessel wall; hence, for the micro-organism in the tonsil to gain access to the blood it must pass through the cervical lymph-nodes, entering the innominate vein via the jugular lymph-trunk. As the lymph-chain of the neck presents a rather formidable barrier against the invasion of micro-organisms, the large majority of patients suffering from microbic infection of the tonsil pass through the disease without any true bacteremia. On the other hand, the absorption by the blood-vessels, of toxins which have been elaborated in the tonsils is a process which accompanies almost every lesion in this organ dependent upon bacteria. The marked systemic depression noticeable in acute tonsillitis is an example.

The relation which the upper respiratory tract holds to pleurisy, as far as the blood route is concerned, can now be quite definitely stated. In a large majority of persons the tonsils act as a source from which bacterial products of greater or less toxicity may gain access to the general system. Further, it can be conceived that there is a degree of toxicity which under ordinary circumstances, as found in the healthy person, would have absolutely no influence. In a person who has been subject to attacks of pleurisy, possibly dependent upon a pulmonary condition, it can readily be conceived that the absorption of toxic substances from the tonsils may be a direct exciting cause to further pleuritic disturbances. In those much rarer cases wherein bacteria themselves gain access to the blood from the tonsils, if the pleura for some reason presents a place of lessened resistance the bacteria may be deposited at this point and there develop.

On the other hand, if the person be in a normal state of health there is no reason why the pleura should suffer from tonsillar infection more than any other portion of the body. This statement is borne out not only by investigation, but also by clinical observation, as shown by reported cases.

We shall now consider the lymphatic infection. A lymphatic infection is naturally propagated via the lymphatic system, passing readily through the vessels, but retarded at each lymph-gland until the functional activity of that node has been overcome. Therefore, concerning lymphatic infection of the pleura from the throat, it is essential, first, to find out whether there normally exist lymphatics connecting the upper deep cervical lymph-glands with the apical parietal pleura; second, if such do not exist, whether diseased processes by cutting off the normal flow of lymph can produce a collateral circulation, whereby lymph from the upper glands of the neck may come in contact with the pleura or its regionary glands.

The lymph-glands of the neck are divided into two groups, the superficial and the deep; the superficial glands drain into the deep ones, generally into the gland which is anatomically the nearest neighbor. The superficial lymph-nodes do not fall within the scope of the present article.

The deep glands situated on each side of the neck comprise from fifteen to thirty nodes on an average, although these figures do not represent the extremes of variation. This group of glands is variously termed the carotid chain, the substernomastoid group, or the deep lateral glands of the neck, and may theoretically and clinically be divided into two groups, although anatomically they seem to make up one continuous mass. They extend from just beneath the ear downward under the sternocleidomastoid muscle, sometimes only as far as the point where the omohyoid crosses the vessels and nerves, but occasionally reaching as far as the junction of the internal jugular and subclavian vein. A gland situated in the notch formed by these veins is for our present purposes of the utmost importance, as will be seen later.

The more superficial division of the deep lateral chain lies posteriorly and is called the external group. The external glands are generally small, and placed in part beneath the posterior border of the sternocleidomastoid, and occasionally extend so far down the anterior border of the trapezius muscle as to come into rather close relation with the supraclavicular glands. The anterior or deeper division of the main group is placed directly over the great vessels of the neck, and is termed the internal jugular group. They are situated beneath the anterior border of the stenocleidomastoid muscle, and when enlarged may be forced anteriorly until some of them appear immediately below the angle of the jaw.

The external glands empty their lymph into the internal jugular group, which in their turn drain one into the other as they descend down the neck, finally emptying into the jugular lymph-trunk. This lymph-trunk empties into the junction of the internal jugular and subclavian veins, and on the left side of the neck is occasionally joined, just before it enters the vein, by the thoracic duct.

The supraclavicular glands occupy the subclavian triangle, and the possibility of connection between this group and the deep cervical glands of the neck is the chief contention in the question of infection from the throat reaching the pleura. The supraclavicular glands receive lymphatics from the posterior part of the hairy scalp and from the muscles of the neck; also, from the skin of the arm and chest, and an efferent branch from the humoral chain of the axillary group. Their efferent duct generally empties into the jugular trunk just before it enters the vein. They are separated from the apical pleura by the axillary plexus of nerves and scalene muscles.

In an interesting series of experiments, Fleiner showed that the supraclavicular glands are regionary to the apex of the parietal pleura. He injected into the pleural cavities of rabbits and dogs both defibrinated blood and India ink, and killed the animals in from five to forty-five minutes afterward. The supraclaviclar nodes contained blood-corpuscles when blood had been injected, and India ink particles when this had been used. He did not, however, trace out the lymph-vessels connecting the pleura and this set of glands.

To make the chain of connection complete from the throat to the pleura there still remains to be seen whether any anastomosis exists between the supraclavicular glands and the external group of the upper deep lateral chain, the lower nodes of which sometimes reach well down along the anterior border of the trapezius.

In 1900, J. A. Grober published quite an elaborate article in which he contended that infections originating in the upper respiratory tract may pass via the cervical lymph-nodes, not only to the pleural apices, but also to the bronchial lymph-nodes. His experiments consisted in injecting a quantity of India ink into the tonsils of a dog and killing the animal six to seven days later. The cervical glands, also the mediastinal and bronchial, the lung and pleura, all showed the presence of India ink particles. The condition, however, was one of extravasation through the connective tissue of the neck rather than lymphatic absorption. Grober's work is not convincing, and he certainly does not demonstrate the presence of lymph-vessels connecting the supraclavicular with the deep cervial lymph-nodes.

Beitzke, in a most thorough and painstaking article, goes over this whole question very thoroughly. He shows that occasionally there does exist a supraclavicular gland which is separated from the pleural apices by the subclavian artery, the branchial plexus, and loose fat tissue, and which has lymphatic connection with the deep glands of the neck. This, however, is a very rare occurrence. Also that another gland, that inconstant node which sometimes occupies the angle made by the junction of the internal jugular vein with the subclavian, can be injected from the tracheo-bronchial glands. These findings would seem to indicate that in certain cases, probably more or less rare, there is direct lymphatic connection between not only the supraclavicular glands and the cervical glands, but also between the tracheobronchial and the cervical.

Beitzke then goes on and gives in detail the results of the post-mortems of forty-five children who showed some tuberculous lesion. Also, he reports a large number of experiments on guinea-pigs and rabbits. In spite of his anatomical findings, he says in his conclusions that there exists no lymph-vessel leading from the chain of cervical lymph-glands to the bronchial glands, and that infection of the lungs from the cervical nodes can only take place through the blood.

My own injections, made by Gerota's method, seem on the whole to substantiate Beitzke's findings. I will describe in some detail two injections, as they show different types of the lymphatic arrangement in the neck.

Case 1. A new-born child. A dissection was made exposing the upper deep cervical nodes. After failing to inject the whole chain from the tonsillar lymph-node, the lower glands were successfully injected from a node situated just above the omohyoid muscle. After hardening in 10 per cent formalin for two days a careful dissection was made. The injection followed the general course downward of the jugular lymph-trunk, which terminated with a saccular dilation at the junction of the subclavian and internal jugular veins. Just before its entrance into the vein the trunk was joined by the thoracic duct. The supraclavicular group, represented by one large node lying on the anterior fibers of the brachial plexus, was not injected. This arrangement of the gland represents a certain type of cases in which the supraclavicular and deep cervical glands are widely separated.

Case 2. A new-born child. The tonsillar lymph-gland was exposed and an injection made into this node without any further dissection. After hardening for several days in a 10 per cent formalin solution, a dissection of the neck was made, and it was seen that the fluid had filled all the glands and the main lymph-trunks of the neck. In this dissection the upper deep chain of cervical lymph-nodes was much more extended than in Case 1, and represents a type in which the different groups are not so easily differentiated. First, there was on the anterior border of the trapezius muscle, well down toward the clavicle, a node which had extensive connections with the rest of the deep cervical chain. It did not communicate directly, however, with the supraclavicular glands, though a very small node with which this gland was connected occupied topographically a position very close to the supraclavicular group. The supraclavicular group was here represented by one fairly large gland, which received the blue injection just as did the glands of the deep chain. The efferent vessel of the supraclaviclar node terminated in the lymph-node situated in the notch formed by the junction of the internal jugular and subclavian veins. This gland also received the terminal efferents of the deep cervical chain. The jugular lymph-trunk was the efferent of this gland and consequently was very short, as the distance between it and the junction of the veins was exceedingly small. This node was separated from the apical pleura only by the common carotid artery and connective tissue, and was therefore in closer proximity to the apex of the pleura than was the supraclavicular node.

Case 2 is very instructive in that it demonstrates the close relation which may exist in certain cases between the supraclavicular glands and the lower lying of the deep cervical chain. While in this case there did not seem to be any connection between the glands on the anterior edge of the trapezius and the supraclavicular glands, they are topographically very closely related, and knowing the variability of lymph-glands and vessels generally it is conceivable that there may be cases in which anastomosis between these two groups exists. Such have been reported by Beitzke. Further, the injection of the supraclavicular gland took place evidently against the current and was due to the node situated in the venous notch obstructing the downward flow, so that the fluid on entering this node probably passed as readily up the efferent of the supraclavicular gland as into the jugular trunk.

From a study of these cases and of the literature it seems that there must be a great variance in the anatomical arrangements not only of the individual nodes of one group, but also of the relations of one group to another. In a large majority of cases the cervical lymph-nodes are separate from the supraclavicular, but it is probable that in isolated cases there exists some lymphatic anastomosis between these two groups. If we accept Fleiner's statement concerning his work, we must believe that the supraclavicular are regionary to the parietal pleura at the apex, and then the chain from tonsil to pleura is completed. In a few injections in which I tried to find lymph-vessels connecting the pleura with the supraclavicular nodes I failed absolutely, but this negative finding does not prove anything.

One other way for an injection to reach the pleural apex from the supraclavicular gland is through the continuity of structure. If the supraclavicular glands should become enlarged, break down, and give rise to a periadenitis, the proximity of the pleura to this group of nodes makes the involvement of the pleura conceivable. The scalene muscles and some loose areolar tissue, and the brachial plexus of nerves, however, separate the glands from the pleura at this point, so that the process must be rather extensive before the pleura can be reached. The node occupying the venous notch and belonging to the deep cervical group comes in closer relation to the apical pleura than the supraclavicular glands, being separated from it only by the carotid artery and its sheath. Enlargement of this node sufficient to cause displacement either anteriorly or posteriorly would bring its capsule into almost direct contact with the pleura. A cellulitis originating from this node might, therefore, quite easily infect the apex of the parietal pleura. We must remember, however, that this gland is the terminating node of the deep chain, and in the large majority of instances the infection does not pass the barriers presented by the glands higher up. Again, it is not a constant condition, its absence being much more common than its presence, so that, considering the question from all sides, it is doubtful whether many cases of pleura infection ever arise directly from it.

It would seem that if the supraclavicular nodes were in the path of infection some light might be obtained on the present question by a careful histological study. This group of nodes, therefore, was taken post-mortem from a number of cases of pulmonary tuberculosis and carefully examined microscopically. Some of the glands were found to be normal, others showed adenitis, and the majority more or less tuberculous change. If it had been found that these glands constantly showed advanced and especially old tuberculous lesions, it would be an indication that they were in the route of the original infection; a constant negative finding would indicate otherwise. The pathological changes, however, occupied such a midway positon as to throw no light whatever upon the etiological importance of these nodes.

During the study of these glands some other interesting conditions were noted, and for this reason the examinations are recorded. In describing the size of the nodes only an arbitrary measure is used. If the gland was considerably less in size than a pea, it is called very small; if about the size of a pea, it is termed small; if about the size of an almond, it is termed moderate; if about the size of a cherry, large; and any larger than a cherry, very large. Both the number and size of the glands varied very markedly in different individuals.

In recording the amount of tuberculous change as found in the glands, I have used the term "slight" when only one, two, or three tubercles were to be found; "moderate" when less than one-third of the gland was involved; and "markedly tuberculous" when more than one-third was involved.

Case 1. Eight glands examined.

Three very small ones showed marked tuberculosis.
One moderately large one showed slight tuberculosis.
Two very small ones showed no tuberculosis.

In all of these six a moderate degree of proliferative adenitis and some anthracosis were seen.

One small gland showed slight tuberculosis, with slight proliferative adenitis.
One small gland was normal.

Case 2. Only one moderately large gland examined. This gland showed moderate tuberculosis, with slight congestion.

Case 3. Two large glands examined.

One showed marked tuberculosis with slight fibrosis, slight congestion, and slight anthracosis. There was also a little hyaline degeneration.

One gland showed slight tuberculosis with a moderate proliferative adenitis, and a moderate amount of hyaline change.

Case 4. Seven glands examined.

One very small gland showed slight tuberculosis with slight proliferative adenitis; also some blood in the lymph-sinuses with slight congestion.

One large gland showed a moderate degree of tuberculosis, with slight proliferative adenitis.

In none of the other glands could any tuberculous changes be found. The remaining five glands were very small, and all showed a slight degree of proliferative adenitis. In four of them there was some blood in the lymph-sinuses.

Case 5. Fourteen glands examined.

One very small gland showed slight tuberculosis with congestion.

Four small glands showed moderate tuberculosis, and one of them was congested.

One moderate-sized gland showed moderate tuberculosis with slight hyaline changes.

One moderate-sized gland showed slight tuberculosis, but also marked fibrosis with anthracosis and congestion.

The remaining seven glands did not show any tuberculous changes. One very small gland was fibrous, and there were some hyaline degeneration and congestion. Three very small glands were normal. One small gland was slightly congested, and two moderate-sized glands were slightly congested.

Case 6. Eight glands examined. None were tuberculous.

One very small gland was normal.

One small gland was normal.

Two small glands showed slight anthracosis, one of them was slightly congested.

One small gland deeply congested, with slight fibrotic changes.

One moderate-sized gland showed congestion and slight fibrosis.

One moderate-sized gland showed slight anthracosis.

One large gland showed slight anthracosis.

Case 7. Four glands examined.

One very small gland showed slight tuberculosis and slight proliferative adenitis.

Two small glands showed slight proliferative adenitis, but no tuberculosis.

One large gland was slightly tuberculous, but otherwise normal.

Case 8. Twelve glands examined.

Six very small glands showed marked tuberculosis.

One very small gland showed slight tuberculosis.

Two very small glands were normal.

Two moderate-sized glands showed moderate tuberculosis, and one of them also slight hyaline degeneration.

One large gland was markedly tuberculous.

Case 9. Two glands only examined.

Both were small; one showed slight tuberculosis, with slight proliferative adenitis and slight anthacosis; the other had a moderate degree of tuberculosis and some anthracosis.

Case 10. Two glands examined.

One very small gland showed slight tuberculosis with slight proliferative adenitis, some fibrosis and some hyaline degeneration.

One small gland showed moderate tuberculosis, with marked fibrosis.

Case 11. Four glands examined.

Three small glands, one was normal, one showed moderate degree of tuberculosis, one was slightly fibroid.

One moderate-sized gland showed a moderate degree of tuberculosis and some fibroid changes.

Case 12. Two glands examined.

Both were large, and showed a moderate degree of anthracosis and some fibroid changes.

One had a suspicion of tubercle.
One had two tubercles, with slight caseation, but no distinct giant cells.

Case 13. Three glands examined.
One very small gland.
Two large glands.

All showed marked tuberculosis, with marked fibrosis and some blood in the lymph-sinuses.

Case 14. Six glands examined.
Three small glands.
Two moderate-sized glands.
One large gland.

No tuberculous changes were found, but there was a marked proliferative adenitis, and the lymph-sinuses contained a large quantity of blood which was being taken up by endothelial phagocytes.

Case 15. Six glands examined.
Five very small glands showed slight proliferative adenitis, only one of them slight tuberculosis.
One small gland showed slight proliferative adenitis and slight tuberculosis.

Case 16. Four glands examined.
All showed marked tuberculosis. Two very small, one large, one very large.

Case 17. Sixteen glands examined.
Nine very small glands were normal.
Two very small glands showed slight tuberculosis, one of them distinct proliferative adenitis with marked hyaline degeneration.
One moderate-sized gland showed slight tuberculosis and some fibrous nodules, which may have been scars of old tubercles.
One moderate-sized gland showed marked tuberculosis, but was otherwise normal.
Three large glands; two showed slight tuberculosis with proliferative adenitis and some hyaline change, and one showed marked tuberculosis with fibrous nodules.

TABULATED CASES.

Anthracosis	6
Blood in lymph-sinuses	3
Adenitis	10
Fibrosis	7
Tuberculosis	15
Hyaline degeneration	5
Congestion	4

TABULATED SUPRACLAVICULAR GLANDS.

	VERY SMALL	SMALL	MODERATE	LARGE	VERY LARGE	TOTAL
Tuberculosis, slight	8	2	3	6	..	19
" moderate	..	7	5	..	..	12
" marked	14	..	1	4	1	20
Proliferative adenitis	21	7	3	4	..	35
Anthracosis	6	2	2	3	..	13
Blood in lymph-sinuses	8	3	2	1	..	14
Fibrosis	5	4	3	2	..	14
Congestion	3	4	5	1	..	13
Hyaline degeneration	4	..	2	3	..	9
Normal	15	3	..	..	..	18

It is unfortunate that in the removal of these glands from the body there was no record kept of the side from which the glands were taken, so that a comparative study with the condition of the lungs and pleura is not possible.

In all, one hundred and one glands were examined, taken from seventeen different bodies.

The importance of the size of the gland in the diagnosis of tuberculosis has, I believe, been overestimated. While the large glands were generally tuberculous, they were not always so, and very small nodes, so small as not to be palpable through the skin, and only found by careful dissection, were frequently the seat of quite extensive disease. The frequency with which proliferation of the endothelium of the lymph-nodes was noticed shows that these glands are subject often to some form of irritation, as would be caused by the presence of a weak toxic substance. This proliferative adenitis was found in 37.6% of all the glands examined. In a large number of the bodies examined there was mixed infection of the lungs, and, as has been suggested by Fleiner, the supraclavicular glands being regionary to the apices of the pleura, it is very probable that the toxins elaborated in the apical lesions pass from thence to the apical pleura, and then find their way to these supraclavicular nodes. In conjunction with this fact it will be noticed that in 12.8% anthracosis was present, and in 13.8% blood was found free in the perilymph-sinuses. Now, as it has been shown, there is in the majority of cases generally no connection between the cervical lymph-nodes proper and the supraclavicular glands, and further it would be quite unlikely that extraneous substances could find their way down through the whole cervical chain into these glands. It cannot be conceived that sufficient dust or coal particles can be absorbed through the skin to give such a marked anthracosis as was found in some of these supraclavicular glands. The only possible source, therefore, for anthracotic material to gain access to this set of glands is from the lungs. The coal-dust passing from the lungs into the pleural cavity, and then being absorbed by the parietal pleura, passes to the regionary glands. In the same way hemorrhage, in cases of phthisis, gives the presence of blood in the lymph-sinuses of these nodes precisely as Fleiner found in his experiments. The presence of anthracosis and free blood in the süpraclaviclar nodes, therefore, is very strong evidence of Fleiner's contention, that the supraclavicular glands receive lymph-vessels from the parietal apical pleura.

SUMMARY.

In summing up briefly the relations between throat infections and diseased conditions of the pleura, according to the data outlined in the present paper, I believe that the following statements are justifiable.

An infecting focus of the upper respiratory tract, and this is most frequently found in the tonsils, may give rise to a pure toxemia or even to a bacteremia. Toxemia is a very frequent condition, and because of its frequency is the most important form of infection coming from the tonsils. Bacteremia originating from the throat is comparatively rare, though a large number of cases have been reported. In either condition, if the pleura is a point of least resistance, as it is in a large majority of phthisical patients, it may very easily be more or less affected by the poison circulating in the blood. The faucial tonsils may present unrecognizable foci of infection, causing in may individuals a mild toxemia, which toxemia, however, is of importance only when the resistance of the individual is below normal.

In exceptional cases there may exist lymphatic anastomosis between the deep cervical lymph-nodes and the supraclavicular glands. It is probable that the supraclavicular glands receive afferent lymph-vessels which drain the parietal apical pleura. Occasionally the efferent of the supraclavicular gland empties into a node situated in the notch formed by the junction of the subclavian and internal jugular veins. This node may also receive the efferent lymph-vessels of the deep cervical lymph-nodes, and is only separated from the pleural apices by the internal carotid artery and its sheath. It is conceivable that lesions of this gland might cause a retrograde current which would carry the infection to the supraclavicular nodes, or a periadenitis of this node might possibly involve the pleura directly through continuity of structure. The necessary links in this infection, however, are so numerous and independent one of the other, that only in very rare cases would it happen that they would all be present so as to form a direct path from the throat to the pleural apex.

BIBLIOGRAPHY.

SECONDARY TUBERCULOSIS OF THE TONSILS.

Seifert: Handbuch der Laryn. u. Rhin., P. Heymann, ii, 714.

Schlenker: Untersuchungen über die Tuberculose der Halsdrüsen, besonders über ihre Beziehungen zur Tuberculose der Tonsillen. Inaug. Diss. Zurich, 1893, u. Virchow's Arch., 1893, cxxxiv.

Schlesinger: Die Tuberculose der Tonsillen bei Kindern. Berlin. Klinik, 1896, xcix.

Walsham: Latent tuberculosis of the tonsil. British Med. Journ., May 7, 1898. Lancet, June 18, 1898.

Ito: Untersuchungen über die im Rachen befindlichen Eingangspforten der Tuberculose. Berlin. klin. Wochen., 1903, xl, 27.

Dmochowsky: Ueber sekundäre Erkrankung der Mandeln und der Balgdrüsen an der Zungenwurzel bei Schwindsüchtigen. Ziegler's Beitr. zur pathol. Anat., x, 481.

Kruckmann: Ueber die Beziehungen der Tuberkulose der Halslymphdrüsen zu der Tonsillen. Virchow's Archiv., cxxxviii, 3.

Strassmann: Ueber Tuberkulose der Tonsillen. Virchow's Archiv., 1884, xcvi, 319.

Ozeki: Die Tuberkulose der Tonsilla palatina. Diss., Würzburg., 1899.

Gourc: Die W. Meyersche Mandel, ein Beiträg zur Bacteriologie der Adenoidgeschwülste. Thèse de Paris, 1897.

Cornil: Sur la tuberculose larvee de trois amygdales. Semaine Médicale, 1895, 223.

Tonsillar Absorption.

Goodale: Absorption of foreign bodies by the human faucial tonsil, especially in regard to the origin of infectious processes. Arch. f. Laryn. u. Rhin., 1897, vii, 90.

Hendelsohn: Absorption by tonsils. Arch. f. Laryn. u. Rhin., 1898, No. 8, 476.

Kayser: Absorption by tonsils. Therap. Woch, v. Schnirer., Oct. 17, 1897.

Etiology of Rheumatism.

C. Wall: A case of serous pleural effusion, following acute rheumatic arthritis and pericarditis. London Lancet, 1905, i, 793.

W. V. Shaw: Acute rheumatic fever and its etiology. Journ. Path. and Bacteriol., 1903-4, ix, 158.

G. W. Webster: Etiology of acute articular rheumatism. Journ. Amer. Med. Assoc., 1903, xl, 80.

R. I. Cole: Etiology of acute articular rheumatism. N. Y. Med. Journ., 1906, lxxxiii, 534.

G. B. Lonstaff: A contribution to the etiology of rheumatism. Tr. Epidemiol. Soc., London, 1905, n. s., xxiv, 33.

E. S. Hawthorne: Acute rheumatism; five cases of apparently direct infection. British Med. Journ., 1903, ii, 1638.

C. Philip: Zur Etiol. des acuten Gelenkrheumatism. Deutsch. Arch. f. kl. Med., 1903, lxxvi, 150.

A. P. Luff: Discussion on rheumatoid arthritis and the morbid conditions which simulate it. British Med. Journ., Oct. 26, 1907.

R. I. Cole: Experimental streptococcus arthritis in relation to the etiology of acute articular rheumatism. Journ. Infectious Diseases, i, 1904, 714.

Tonsillitis and Rheumatism.

H. Suchannek: Relations of angina to acute rheumatism of the joints. Bresgen's Sammlung zwangl. Abhandl., u. s. w. Heft 1.

Joseph Meitus: Tonsillitis and its relation to rheumatism. Cincinnati Lancet-Clinic, March, 1896.

F. A. Packard: Infection through the tonsils, especially in connection with acute articular rheumatism. Phila. Med. Journ., 1900, 957.

St. Clair Thompson: Rheumatic fever in relation to the throat. Laryngoscope, x—xi, 1901, 23.

A. Jacobi: The pharynx and nose in rheumatism. N. Y. Med. News, Dec. 15, 1900.

A. Stengel: Articular rheumatism. Amer. Journ. Med. Sci., n. s., cxxv, 1903, 412.

R. T. Turner: Acute rheumatism. Three cases of apparently direct infection. British Med. Journ., 1903, ii, 311.

T. McCrae: Acute articular rheumatism. Journ. Amer. Med. Assoc., xl, 1903, 211.

Chas. F. Kuhn: Tonsillitis and its complications. The Physician and Surgeon, xxviii, 1906, 164.

P. W. Williams: The throat as a source of systemic infection in acute rheumatism. Bristol Med. Chir. Journ., xxii, 1904, 215.

A. C. Miller: Tonsillitis as a prodrome to acute articular rheumatism. Columbus Medical Journal, April, 1904.

Editorial: The tonsils as portals of rheumatic infection. Journ. Amer. Med. Association, Jan. 19, 1901.

Gurich: Ueber die Beziehungen zwischen Mandelerkrankungen und dem akuten Gelenk-rheumatismus. Münch. med. Wochens., 1905.

E. Fletcher Ingalls: The relation of tonsillitis to rheumatism. Laryngoscope, xvii, 1907, 712.

J. Phillips: Acute articular rheumatism. Cleveland Medical Journal, December, 1906.

G. Epstein: Angina, erythema exsudativum multiforme, pleuritis exsudativa dextra. Wien. kl. Rundschau, 1905, xix, 73.

Hammerschmidt: Ueber den Zusammenhang der Mandelentzündung mit anderer Krankheiten. Deutsch. Militarärzt. Ztsch., xxxii, 1903, 257.

Menzer: (Quoted by Hammerschmidt.)

Isaac Adler: Remarks on some general infection through the tonsils. N. Y. Med. Journ., 1906, lxxxiii, 641.

D. J. M. Miller: Infection through the tonsils. N. Y. Med. Journ., 1905, lxxxi, 1036.

Bruck: Purpura rheumatica und angina. Berl. kl. Wochensch., 1900, xlv, 1005.

Trosseau: Clinique médicale de l'Hôtel Dieu, 1895.

Lagranère and Pipounier: Essay sur les angines rheumatism et goutteuses. Thèse. Paris, 1876.

Caesar Boeck: Referat in Schmidt's Jahrbücher, 1882, xix, S. 33.

Lasègue: Archives générales de Médicine, 1880.

H. Brown: Brit. Med. Journ., Sept. 14, 1889.

Stephanides: Prag. med. Wochensch., No. 50, 1885.

Whipham: Report of the Investigation Committee of the British Medical Association. Brit. Med. Journ., Feb. 25, 1888.

Roos: Ueber rheumatische Angina. Berlin. klin. Wochensch., xxv, xxvi. 1894.

F. Peltesohn: Ueber Angina und Rheumatismus. Archiv für Laryngol. und Rhinol., 1897-98.

Gurich: Der Gelenkrheumatismus, sein tonsillarer Ursprung und seine tonsillare Heilung. Published by Max Woywod, Breslau, 1905.

J. L. Goodale: Some considerations relative to systemic infection through the tonsillar ring. The American Laryngological Association, 1907.

E. Maragliano: Importenza della tonsilla per la etiologia della poliartrite reumatica. Gazett. degli ospedali., xxx, Marz 10, 1901.

TONSILLITIS AND GENERAL INFECTION.

E. Lexer: The mucous membrane of the throat as a port of entrance for pyogenic infection. Arch. f. kl. Chir., Bd. 54, 1897, 736.

Piera: Die Drusen des Waldeyer'schen Schlundringes, special die Gaumentonsillen betrachetet als Eingangspforte für Micro-organism. Arch. Ital. d. Laringol., April 1, 1900.

M. Otten: Staphylococcusmycosis. Deutsch. Arch. f. kl. Med., xc, July, 1907.

G. A. Woodcock: Pyemia and phlebitis complicating tonsillitis, with report of case. Iowa Med. Journ., Des Moines, x. 1904, 256.

Stengel, White and Evans: Cryptogenetic streptococcus infection with persistent cutaneous eruption, enlargement of the lymphatic glands and fever, suggesting syphilis. University of Penna. Med. Bul., xix, 1906, 217.

Tishutkin: Case of deadly poisoning, caused by virulent micrococci in the tonsil. Wratsch, No. 9, 1897.

C. E. Edson: Case of septicemia treated with normal salt solution. Boston Med. and Surg. Journ., July, 1899.

W. L. Wainwright: Tonsillitis accompanied by rapidly fatal pyemia. Brit. Med. Journ., ii, 1897, 651.

I. Owen: Case of blood poisoning from tonsillitis; comparative results obtained from two varieties of antistreptococcic serum and from perchlorid of iron. London Lancet, Oct. 15, 1904.

F. Jessen: Ueber die Tonsillen als Eingangspforte für schwere Allegemeininfectionen. Münch. med. Wochensch., No. 23, 1898, 709.

Buschke: The tonsils as the entrance gates for pyogenic microorganisms. Deutsch. Ztschrift. f. Chir., xxxviii, 1894, 441.

Isaac Adler: Remarks on some general infection through the tonsils. N. Y. Med. Journ., lxxxiii, 1906, 641.

W. S. Anderson: Infection through the tonsils. N. Y. Med. Journ. lxxxi, 1905, 852.

A. Jacobi: Infection by way of the tonsil. N. Y. Med. Journ., lxxxiv, 1906, 360.

Trautmann: Bedeutung der nasalen Athmung in ihrer Schutz und Heilwirkung auf Krankheiten. Monatsch. f. Ohrenheilkunde. Berlin, xxxvi, 1902, 530.

Obtulowicz: Intern. Centralb. f. Laryngol., Bd. v, S. 213.

Buss: Ueber bakterielle, haemorrhagische Infectionen des Menschen. Wiener med. Wochen., No. 36 u. ff. 1892.

Babes: Ibid.

Dubost: Sur les complications septicémiques et pyohémiques des angines aigues non dipthèriques. Thèse de Paris. 1896.

Tonsillitis and Endocarditis.

J. S. Ross: The rôle played by the tonsils in organismal diseases. Med. Press and Circ., London, 1906, n. s. lxxxi, 333.

Charrin: Staphylococcic endocarditis. Located on the pulmonary valve, originating from the tonsils. Semaine Méd., March 14, 1896.

E. Mayer: The tonsils as portals of infection. Journ. Amer. Med. Assoc., Dec. 2, 1899.

F. A. Packard: Endocarditis of tonsillar origin. Boston Med. and Surg. Journ., May 25.

Tonsillitis and Nephritis.

J. L. Morse: Tonsillitis a cause of acute nephritis. Arch. of Pedr., 1904, 337.

Hammerschmidt: Ueber den Zusammenhang der Mandelentzündung mit anderer Krankheiten. Deutsch. Militarärzt. Ztsch., xxxii, 1903, 257.

I. Adler: Remarks on some general infection through the tonsils. N. Y. Med. Journ., lxxxiii, 1906, 641.

A. Thouvenet: Tonsillar nephritis. N. Y. Med. Journ., May 5, 1894.

Baduce: La settimana medicale 1896. Deutsch. Med., No. 34, 1896.

Jeppe: Internat. Centralb. f. Laryng., Bd. vii, S. 438.

Czajkowski: Ueber Complicationen der idiopathischen acuten Pharyngitis. Internat. Centralbl. f. Laryngol., Bd. ix, S. 21.

TONSILLITIS AND BRONCHITIS.

Hammerschmidt: Ueber den Zusammenhang der Mandelentzündung mit anderer Krankheiten. Deutsch. Militarärzt. Ztsch., xxxii, 1903, 257.

TONSILLITIS AND PNEUMONIA.

Hammerschmidt: Ueber den Zusammenhang der Mandelentzündung mit anderer Krankheiten. Deutsch. Militarärzt. Ztsch., xxxii, 1903, 257

Adler: Remarks on some general infection through the tonsils. N. Y. Med. Journ., 1906, lxxxiii, 641.

W. Wallace: Septic pneumonia following inflammation of the throat. Lancet, Feb. 15, 1896.

TONSILLITIS AND PLEURISY.

A. Heddaeus: Tonsillitis acuta durch Staphylococcus pyogenes aureus. Pleuritis exudativa metastat. Diplococcuspneumonia. Thoracotomie, sepsis exitus. München. med. Wochen., No. 18, 1897, 467.

Huguenin: Ueber kryptogenetische Pleuritis. Correspondenzblatt f. Schweizer Aerzte., No. 3, 1893.

Gouguenheim: Annales des maladies de l'oreille, x, October, 1896.

TONSILLITIS AND PHLEBITIS.

J. Trumbull: Infective phlebitis as a sequel to follicular tonsillitis. N. Y. Med. Rec., Aug. 9, 1890.

Wagner: München. med. Wochen., No. 9, 1892.

Hager: Intern. Centralb. f. Laryngol., Bd. v, S. 212.

Sahli: Deutsches Archiv. f. klin. Med., Bd. li, 1893.

TONSILLITIS AND MENINGITIS.

Hammerschmidt: Ueber den Zusammenhang der Mandelentzündung mit anderer Krankheiten. Deutsch. Militarärzt. Ztsch., xxxii, 1903, 257.

Perigord: Ein Fall von infectioser Angina. Limousin Médical. Aout, 1893.

DeGuelder: Streptokokken-Angina mit Meningitis. Annal de la Soc. Med. Chir. d'Anvers., Mai and Juni, 1901.

Tonsillitis and Tenosynoitis.

Forchheimer: Diseases from tonsillar infection. Arch. of Pedr., xix, 1902, 656.

Freeman: Ibid.

E. W. Mitchell: Amygdalitis, followed by appendicitis, nephritis and endocarditis. Arch. of Ped., xx, 1903, 182.

Tonsillitis and Skin Lesions.

Le Gender: Amygdalitis and cutaneous eruptions. N. Y. Med. Journ., April 23, 1892.

Leven: Fall von Sclerodermie nach Angina. Dermat. Centralb., v, 1902, 4.

Legendu and Claisse: Purpura and erythema papulo-nodosum, occurring during an attack of streptococcic tonsillitis. Semaine Méd., 1892, 10.

Bruck: Purpura rheumatica and angina. Berl. kl. Wochen., xlv, 1005.

Tonsillitis and Parotitis.

Hammerschmidt: Ueber den Zusammenhang der Mandelentzündung mit anderer Krankheiten. Deutsch. Militarärzt. Ztsch., xxxii, 1903, 257.

Tonsillitis and Eye Lesions.

Vergely: Krankenvorstellung, acute Mandelentzündung; drohende Phlegmone der Orbita. Journ. d. med. d. Bordeaux, xxix, July 19, 1896.

Tonsillitis and Jaundice.

Forchheimer: Diseases from tonsillar infection. Arch. of Ped., xix, 1902, 656.

Tonsillitis and Tenosynovitis.

Tracy: Acute tenosynovitis following tonsillitis. Boston Med. and Surg. Journ., July 25, 1907.

On the Lymphatic Infection of the Pleura.

Beitzke, H.: Ueber den Weg der Tuberkelbazillen von den Mund. und Rachhöhlen, mit besonderer Berucksichtigung der Verhältnisse beim Kinde. Virchow's Archiv. f. p. Anat., Berl., clxxxiv, 1906, 1.

Bunting, T. L.: The histology of lymph glands; the general structure, the reticulum, and the germ centres. J. Anat. and Phys., London, xxxix, 1904-5, 55.

Buttersack: Wie erfolgt die Infection der Lungen. Ztschr. f. kl. Med., 29, 1896, 411.

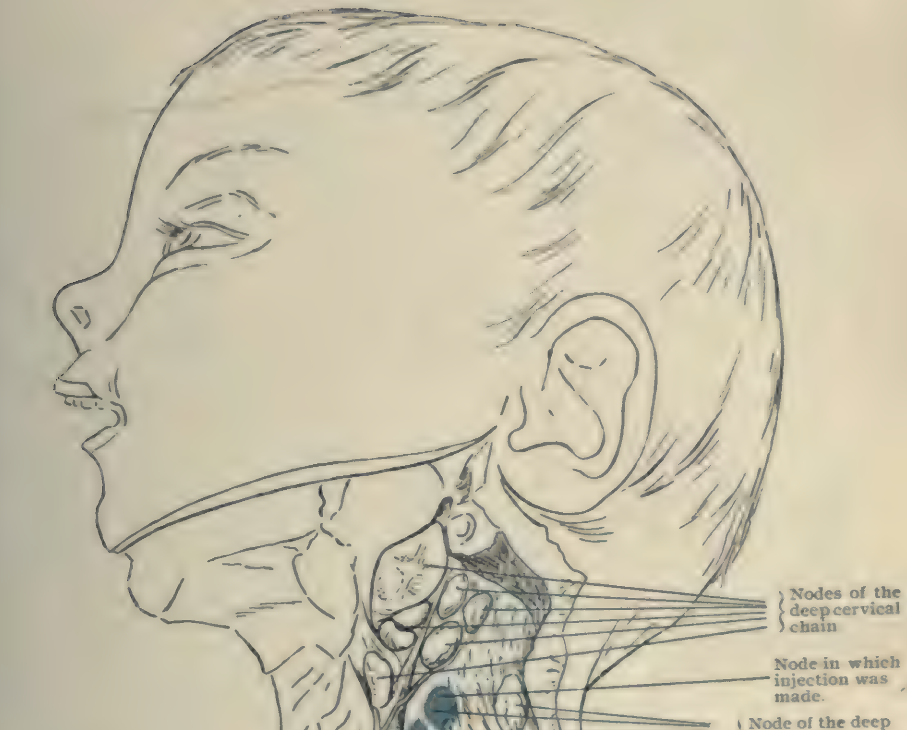
Fleiner: Ueber die Resorption Corpusculäres Elemente durch Lungen und Pleura. Virch. Archiv., Bd. 112, 1888, S. 97.

Flicker: Ueber die Aufnahme von Bakterien durch den Respirationsapparat. Arch. f. Hyg., Bd., 53, S. 50.

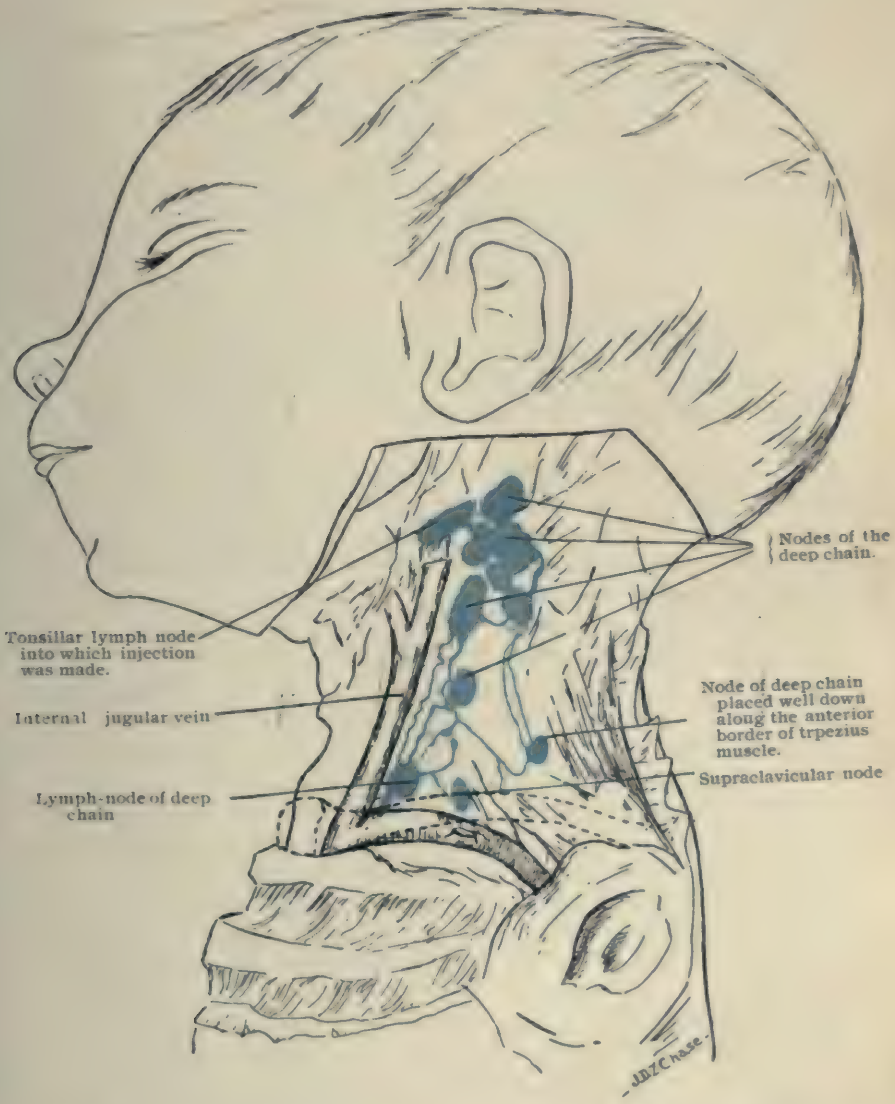
Gerota: Zur Technik der Lymphgefassinjektion. Anat. Anzeiger, Bd. 12, 1896, S. 216.

Grober: Die Infektionswege der Pleura. Deut. Arch. f. kl. Med., Bd. 68, 1900, S. 296.

Von Ruck: The relation of pleurisy to tuberculosis. N. Y. Med. Journ., lxxxii, 1905, 695.


Nodes of the deep cervical chain
Node in which injection was made.
Node of the deep cervical chain
Internal juglar
Brachial plexus
Supraclavicular node
Thoracic duct
Sacular dilation of jugular lymph trunk
Subclavian vein
JDZ Chase

CASE I. Injection of cervical lymph-glands and vessels showing no connection between supraclavicular and deep lateral glands. (See page 189.)


Nodes of the deep chain.
Tonsillar lymph node into which injection was made.
Internal jugular vein
Node of deep chain placed well down along the anterior border of trpezius muscle.
Supraclavicular node
Lymph-node of deep chain
JDZChase

CASE II. Injection of cervical lymph-glands from the tonsillar lymph gland. The efferent vessel of the supraclavicular gland empties into the lowermost node of the deep lateral chain.

XXXV.

THE MORPHOLOGY OF THE TURBINALS.

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The turbinal bones may be classified, in a general way, according to their function, into respiratory turbinals and olfactory turbinals. They are also classified as maxillary, nasal and ethmoidal turbinals, according to the bones which support them.

Fish. The nasal organs of all fish, except Dipnoi (lung fishes), are used only for olfaction, and so contain only olfactory turbinals or tissue, which corresponds to the olfactory turbinals in other vertebrates. Respiration is, of course, carried on entirely through the gills. In the whitefish of the Great Lakes (Coregonus Clupeiformis) the nasal organs are two irregularly spherical-shaped cavities, separated from each other by a thick septum. They are situated in the anterior part of the head. Each cavity opens externally through two orifices in its external lateral wall, but does not communicate with the mouth. The posterior orifice is supplied with a valve, which when opened permits the water to circulate through the nasal cavity.

On the internal lateral wall of each cavity there are twelve finger-like projections or lamellae radiating from a common center, which are covered with olfactory epithelium in which the filaments of the olfactory nerve end (Fig. A), and they constitute the olfactory turbinals of the fish.

These lamellae, like the turbinals in other vertebrates, increase the expanse of mucous membrane exposed within a limited space, and so increase the acuteness of the sense of smell.

Reptiles. The character and structure of the nose in reptiles is modified by the change in the method of respiration. The nasal fossae become differentiated into a respiratory and an olfactory portion. The pterygopalatine arch is well developed and the shelf-like palatine processes of the superior maxillae unite with the palatine bones and form a secondary roof to the mouth. The fossae are thus prolonged backward, below the brain, and open into the nasopharynx.

The Crocodilia, to which order the alligator belongs, are the most highly developed of the reptiles, and their nasal organs are much more complicated than in the fish.

In the alligator, the two nasal cavities are separated in the median line by a rather thick, smooth septum (Figs. C and D, 1). Each cavity opens externally through a small elliptical orifice in the extreme anterior superior surface of the snout. The anterior half of each nasal cavity is the respiratory portion of the nose. There is a round ridge, projecting downward from the roof of this part of the nose (Figs. B and C, 4), which is a true turbinal and is homologous with the inferior or respiratory turbinal in man. The posterior half of each nasal cavity is subdivided into two superimposed cavities of which the upper one is much the larger and may be designated as the olfactory region, for it contains the olfactory turbinals and mucous membrane.

The inferior cavity is simply a round tube, which conducts the inspired air backward from the respiratory portion of the nose into the nasopharynx (Figs. B and D, 8).

The olfactory turbinal is a curved process attached to the roof of the olfactory portion of the nose (Figs. B and D, 2). The posterior end is simply a ridge projecting downward into the nasal fossa, but along the middle portion the turbinal increases in size and its inferior border curves outward toward the external wall of the nose. The anterior portion is much larger than the rest of the turbinal and contains a distinct cell. The olfactory lobe of the brain extends forward and ends just behind the posterior portion of this turbinal, and the olfactory nerves are distributed over it and to the surrounding mucous membrane (Fig. B, 6). The general characteristics of this turbinal correspond quite closely to the middle turbinal, its homolog in man.

Birds. In most birds the anterior portions of the nasal fossae communicate with each other, the septum being incomplete (Fig. E, 1). Posteriorly both fossae open together into the mouth through an elliptical fissure in the middle of the posterior part of the palate. The turbinals are more highly developed than they are in fish or reptiles.

The Loon (Colymbus) is a web-footed swimming bird, which lives in the cold waters of the nothern hemisphere. It has a long beak and the nasal fossae extend forward in it about one-half of its length. Each fossa contains three turbinals. The most anterior one is attached to the roof and external lateral wall of the fossa by a slender pedicle, which gives off five distinct branches. It is called the vestibular turbinal on account of its location in the vestibule or extreme anterior part of the nose, but it is covered with stratified pavement epithelium and does not correspond to any of the turbinals in man (Fig. E, 2).

Just above the posterior end of the vestibular turbinal, a second turbinal begins and extends backward through the rest of the fossa. Its anterior part is attached to the roof of the nasal cavity and its inferior border is curved outward. As the turbinal extends backward, its attachment shifts from the superior to the lateral wall and it increases in size and becomes more and more rolled, so that in its posterior part it is decidedly scroll-shaped and occupies the greater part of the nasal fossa (Figs. F and G, 3). This turbinal is the homolog of the inferior or maxillary turbinal in man. and it is covered with ciliated epithelium.

The third turbinal is situated in the extreme posterior superior part of the nasal fossa. It is a simple ridge-like structure, supported by a broad base, which is attached to the external lateral wall. Its anterior end contains a small closed cavity (Fig. G, 4). The olfactory mucous membrane is distributed over this turbinal and the roof of the nasal fossa in this region. This turbinal corresponds to the ethmoidal turbinal in man.

MAMMALS. The turbinal structures in mammals exhibit great variations. In microsmatic mammals (animals with an acute sense of smell) the turbinals attain their highest degree of development and are very complicated structures.

The turbinals in the American panther (Felis concolor) are a good example of this high degree of development. The nasal fossae are separated from each other by a thin, straight septum, which unites with the cribriform plate posteriorly. Inferiorly the septum is formed by the vomer and presphenoid, and superiorly by the descending plates of the frontal and nasal bones. Along the articulation of the perpendicular plate of the ethmoid with these descending plates, a lateral process extends out at right angles into each fossa and supports a considerable portion of the olfactory turbinals and partially subdivides the olfactory portion of the nose into two superimposed cavities (Fig. J, 1).

The maxillary turbinal is in the anterior inferior part of the nasal fossa (Figs. H and I, 2). It springs from the superior maxillary bone by a slender pedicle, which extends out into the fossa, nearly parallel to the floor of the nose, and divides into two branches. The superior branch curves upward, making one complete coil. The inferior branch curves downward and makes two turns within itself. The whole structure is a typical example of the double coiled turbinal, which is present in many mammals. It is the homolog of the inferior turbinal in man.

The naso-turbinal is a rather simple structure, which projects downward from the nasal bone and overlaps the maxillary turbinal and the anterior part of the ethmoidal turbinals (Fig. H, 6). Its homolog in man is the ager nasi, a small bony ridge anterior to the middle turbinal.

The ethmoidal or olfactory turbinals are very complex structures. There are six of these turbinals in each fossa. They radiate from the convex surface of the cribriform plate, and the extent of their surface is tremendously increased by almost innumerable folds and branches, which make an intricate labyrinth occupying the posterior part of each nasal fossa and constitute the olfactory portion of the nose (Figs. H, I and J, 3). Branches from these turbinals extend into all of the accessory sinuses.

The posterior portion of each nasal fossa is subdivided into two superimposed cavities by the lateral wings of the vomer (as it is in the alligator). The superior cavity is the olfactory portion of the nose, and contains most of the olfactory turbinals. The inferior cavity connects the respiratory portion of the nose with the nasopharynx (Figs. H and J, 4).

Apes. The relative position of all of the turbinals is changed in apes and in man. Especially in man, the tremendous development of the fore-brain pushes the ethmoidal and frontal bones forward and the nose appears to recede into the head. The cribriform plate, in its new position, forms the greater part of the roof of the nose and the ethmoidal turbinals are situated more directly above the maxillary turbinals.

The turbinals in the gibbon or long-armed ape (Hylobates) are comparatively simple structures, but they are less degenerated than those of some other apes. The maxillary turbinal has a double coil, but the superior coil is much smaller than the inferior one and thus shows the tendency toward degeneration and disappearance of this part of the turbinal in the higher animals (Figs. K and L, 2). The ethmoidal turbinals show a much more marked degeneration. There are two in each fossa and their position and general appearance is the same as the ethmoidal turbinals in man (Figs. K and L, 3 and 4). They have entirely lost the numerous fine branches which are present in some of the lower animals, and their number has decreased from five to two. Immediately in front of the ethmoidal turbinals, on the external lateral wall of each nasal fossa, there is a bony ridge, the ager nasi, which is the rudiment of the nasal turbinal (Fig. K, 6).

MAN. The characteristics of the turbinals in man are too well known to need a description here. They are comparatively rudimentary structures, which have reverted to the more primitive types present in the lower vertebrates.

To recapitulate briefly, the turbinal structures of fish (except dipnoi) are used only for olfaction, and are simply ridges covered by olfactory mucous membrane. In reptiles, owing to the changed manner of respiration, the nasal organ functionates both in respiration and olfaction, and respiratory and olfactory turbinals are found. They are, however, quite simple structures. In birds the importance of the respiratory function of the nose increases and the respiratory turbinals show a high degree of development. The expanse of respiratory mucous membrane is very largely increased by the coiled structures of the turbinals. The olfactory turbinals are simple ridge-like structures.

In microsmatic mammals all of the turbinals exhibit a very high degree of development. Their numerous fine branches and coils enormously increase the amount of mucous membrane exposed within a comparatively small space and thus increases the efficiency of the nasal organs. In apes all of the turbinals have degenerated or reverted to some of the more primitive types. In man the turbinals are quite similar to the turbinals of the ape. They are all rather simple structures. Rudiments of the fourth and fifth ethmoidal turbinals are sometimes found. The ager nasi is the rudiment of the nasal turbinal. Such rudiments are much more frequently present in the embryo than in the adult.

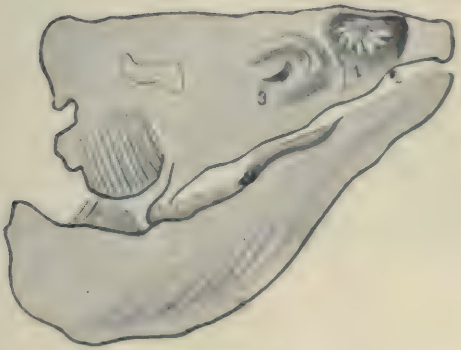

FIGURE A.

Lake Whitefish (Coregonus Clupeiformis). Longitudinal section through the right nasal fossa looking toward the septum.

1. Nasal fossa, showing the olfactory lamellae radiating from a common center on the internal lateral wall.
2. The mouth.
3. The eye.

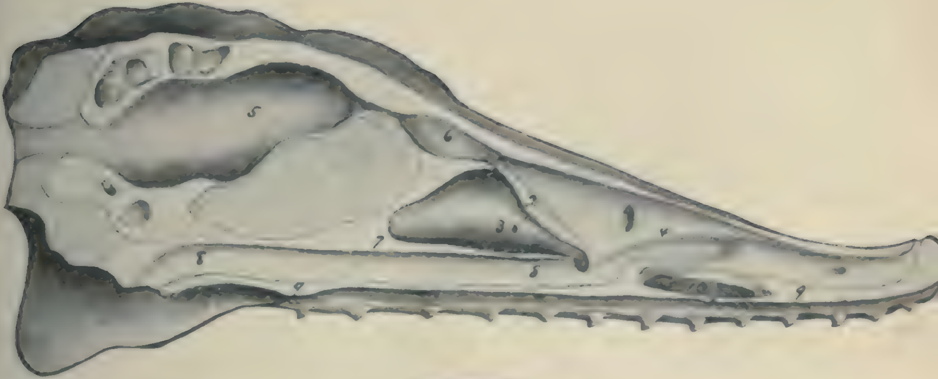

FIGURE B.

ALLIGATOR. Longitudinal section, showing the left nasal fossa.

2. Olfactory turbinal.
3. Pseudo turbinal.
4. Maxillary turbinal.
5. Brain cavity.
6. Olfactory lobe of brain.
7. Lateral wing of vomer.
8. Respiratory portion of the posterior part of the nose.
9. Palate.
10. Maxillary cavity.

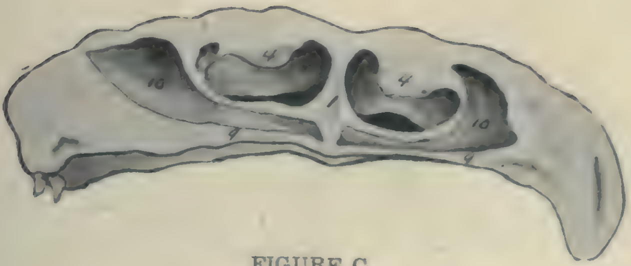

FIGURE C.

ALLIGATOR. Transverse section through both nasal fossae, in the anterior or respiratory portion of the nose, looking anteriorly.

1. Nasal septum.
4. Maxillary turbinal.
9. Palate.
10. Maxillary cavity.

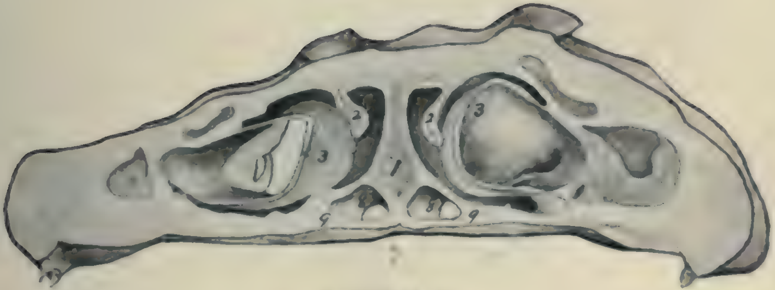

FIGURE D.

Alligator. Transverse section through the olfactory portion of both nasal fossae, looking posteriorly.

1. Nasal septum.
2. Olfactory turbinal.
3. Pseudo-turbinal.
8. Respiratory tract.
9. Palate.

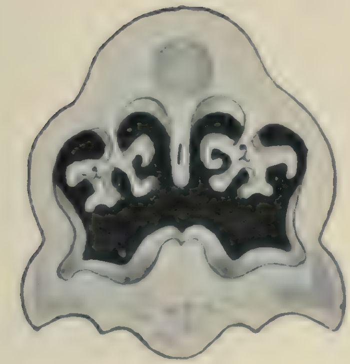

FIGURE E.

LOON. Transverse section through the anterior part of both nasal fossae, looking posteriorly (twice natural size).

1. Nasal septum. The free communication between the two nasal fossae in this part of the nose is shown.
2. Vestibular turbinal.

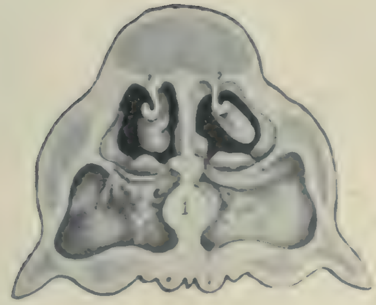

FIGURE F.

LOON. Transverse section through the middle portion of both nasal fossae, looking posteriorly (twice natural size).

1. Nasal septum.
3. Maxillary turbinal.

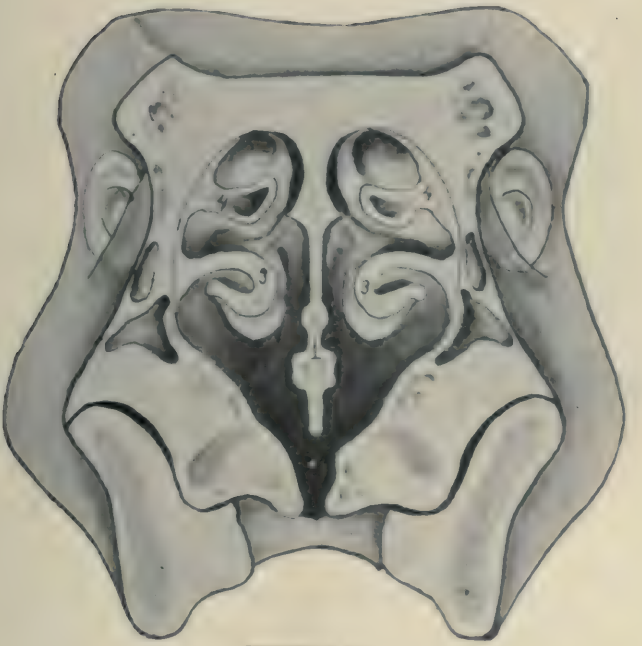

FIGURE G.

Loon. Transverse section through the posterior portion of both nasal fossae, looking posteriorly (twice natural size).

1. Nasal septum.
3. Maxillary turbinal.
4. Olfactory turbinal.

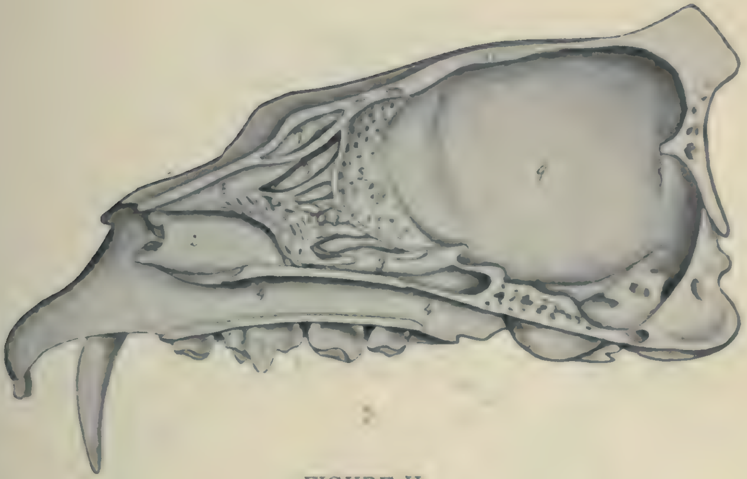

FIGURE H.

Panther. Section showing the right nasal fossa of the American Panther (one-half natural size).

2. Maxillary turbinal.
3. Ethmoidal turbinals.
4. Respiratory portion of the nose separated from the olfactory portion by the lateral wing of the vomer.
6. Nasal turbinal.
7. Ethmoidal turbinal in frontal sinus.
8. Cribriform plate.
9. Brain cavity.
10. Sphenoidal sinus.

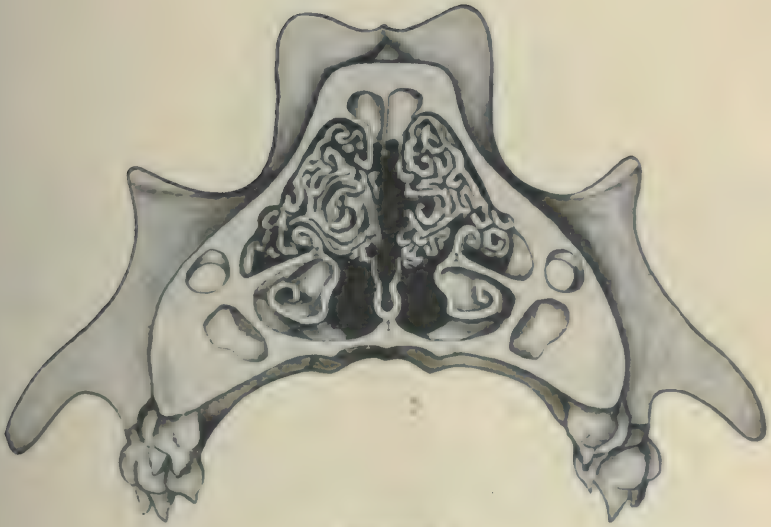

FIGURE I.

Panther. Transverse section through the anterior part of both nasal fossae, looking posteriorly.

1. Nasal septum.
2. Maxillary turbinal.
3. Ethmoidal turbinals.

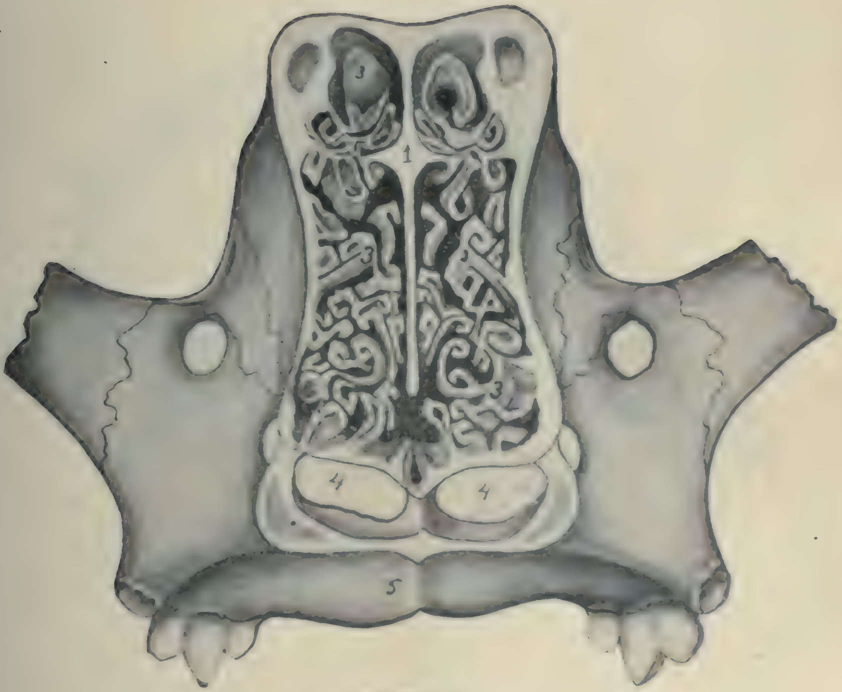

FIGURE J.

Panther. Transverse section through the middle portion of both nasal fossae, looking anteriorly.

1. Nasal septum.
3. Ethmoidal turbinals.
4. Respiratory portion of the nose separated from the olfactory portion by the lateral wing of the vomer.
5. Palate.

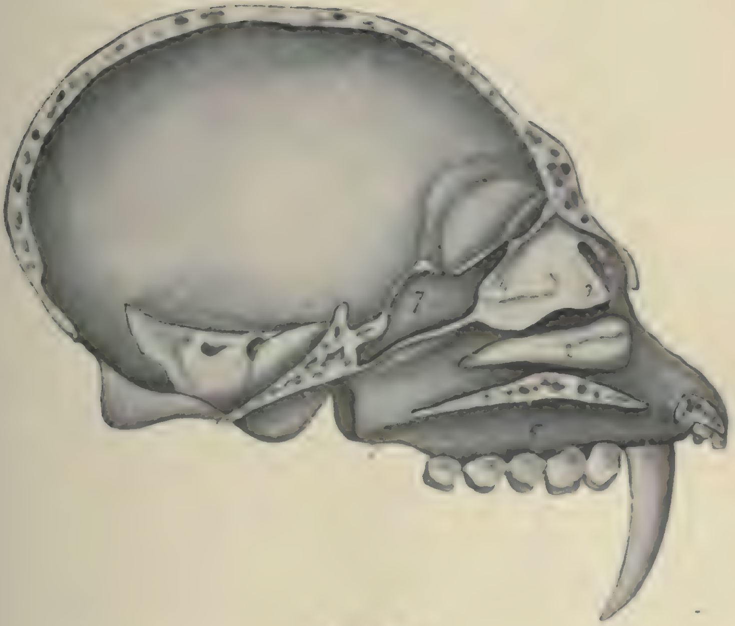

FIGURE K.

Ape. Longitudinal section showing the left nasal fossa.

2. Maxillary turbinal.
3. Ethmoidal turbinal.
4. Superior ethmoidal turbinal.
5. Palate.
6. Nasal turbinal.
7. Sphenoidal sinus.

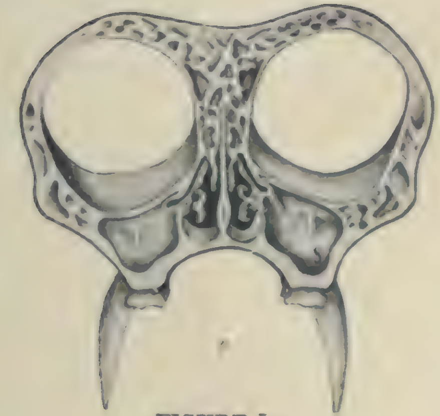

FIGURE L.

Ape. Transverse section through both nasal fossae, looking anteriorly.

1. Nasal septum.
2. Maxillary turbinal.
3. Ethmoidal turbinal.
8. Maxillary sinus.

XXXVI.

GIANT TUMORS OF THE LARYNX.

By Prof. F. Massei,

NAPLES.

From a total of nearly 600 cases in my personal statistics, embracing 38 years of practice, I have selected 10 cases of tumor of the larynx which not only attained extraordinary size, but also presented special characteristics, giving them a certain value pathologically and clinically.

I shall describe them briefly and permit myself a few remarks in the form of conclusions.

The first case, seen in 1872, was that of a man, aged 48, of slight build, who, since his seventh year, had a nasal voice. In his later years he slept involuntarily several times a day. Respiration was noisy, but deglutition was normally effected.

Laryngoscopic Examination.—A tumor almost three centimeters in diameter, round, smooth, pale red in color, of firmly elastic consistence, was implanted upon half the laryngeal surface of the epiglottis, and had invaded the vestibule. Its base was fixed.

Three years after my first examination the tumor had increased in volume and the dyspnea was worse. I proposed tracheotomy and an attempt at ablation of the tumor with a galvano-cautery snare, but the patient refused all treatment and was lost sight of.

From a clinical point of view I shall admit that this was a fibroma. As may be seen in the drawing, it seems much larger than the one shown by Mackenzie in his classic treatise, but it is much smaller than the one described by Rokitansky, which reached the size of a pigeon's egg.[1]

The second case is that of a man, aged 50, of weak constitution,[2] who, for two years, had intermittent hoarseness,

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(1) This case was published in my "Les Neoplasmes du Larynx."—See Italian Archives of Laryngology, 1884-85.

(2) Francesco de Feo, of Giffoni Valle-Piana, hairdresser. See No. 285 of my private register.

then becoming constant. When I saw him the first time, in June, 1906, he was aphonic and dyspneic.

Laryngologic Examination.—Springing from the right ventricle of Morgagni is a large mass (shown in Fig. 2) protruding into the glottis. It has a wide base, and in color and appearance resembles an hypertrophied tonsil hanging down in the hypoglottic region.

A part of the tumor has its origin on the right cord.

August 9, 1906, I excised it with forceps and after having removed almost all the growth I discovered that it also took origin from the epiglottic petiole.

Four months later, when I saw him again, he was entirely well.

Microscopic examination by Prof. Martuscelli showed that it was a cystic fibroma, as the cut indicates. (Fig. 11.)

It is known with what facility similar tumors sometimes attain considerable size, and for this reason I will describe briefly another case of this sort:

Case 3.—Man, 46 years old,[1] tall, but weakly, and an epileptic. Seen at my hospital clinic in January, 1898.

Three years previously he had caught cold, but he states that the breathlessness which bothered him had recently increased to the point where suffocation threatened.

Laryngoscopic Examination.—An enormous polyp of the left vocal cord, pale, glossy, smooth and mobile, resembling a nasal polyp, occupies the entire laryngeal cavity. (See Fig. 3.)

I decided to operate per vias naturales, but next day the tumor had increased so much in size and the dyspnea had so augmented that I was obliged to recommend a preliminary tracheotomy. Prof. Salvia did this two days later.

Seventeen days afterward, with forceps, I removed the tumor entire, which had shrunk to the size of a bean.

A month later the canula was taken out. The tumor has not recurred.

Prof. Martuscelli, who made the microscopic examination, confirmed my diagnosis of cystic fibroma. (See Fig. 12.)

The striking feature in this case was the rapid increase of size (due perhaps to hemorrhage into the lacunar spaces) and

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(1) Raffaele Tramontano, of Capua. No. 106, in my clinical register A.

then the diminution after tracheotomy. Like the preceding case, it shows what a volume may be attained and how many surprises a cystic fibroma of the larynx, usually of small size, may give us.

Case 4.—A little girl of 4 years[1] was tracheotomized in 1904 by Dr. Cardoso, of Tunis, for a violent attack of laryngeal stenosis and was sent to me at Naples eighteen months later, July 14, 1908, for decanulation.

Autoscopy and mirror examination showed a large mass, of cauliflower form, the size of a nut, pale red in color, of irregular shape and surface, which appeared to issue from the esophageal aditus and to be supported by the arytenoid cartilages, which were hidden. The laryngeal vestibule was also involved. (See Fig. 4.)

Any attempt to close or remove the canula stifled the child. On the other hand, intubation was surprisingly successful, and completely reestablished respiration.

I continued the intubation at intervals, alternating by tearing away the papilloma with forceps, either per vias naturales or through the tracheal fistula. Papillomata could also be seen in the trachea, and although the principal mass was gone, the tube would not go any further down in the larynx. By autoscopy I made out a great number of buds springing from the interarytenoid mucosa.

After a year of patient treatment there were only traces of the growth. The canula could be closed for some time. Unfortunately, the little girl was obliged to go back to Tunis to be treated for a tubercular arthritis of the elbow.

Histologic examination by Prof. Martuscelli (see Fig. 13) confirmed the diagnosis of papilloma, no tubercles being found.

In this case, interesting from the viewpoint of extension of the growths, and having an analogy to Bethan Robinson's,[1] I did not want to perform laryngo-fissure, partly through fear of recurrence, which renders all radical intervention illusory, and partly through fear of an invasion of the trachea by papillomata. My patience might have been crowned with success and a cure obtained by a continuation of treatment.

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(1) Elena Buonocore, of Tunis. No. 282 of my register.

(1) Proceedings of the London Laryngological Society, Vol XIV, March 8, 1907.

Case 5.—B. R.,[1] 50 years old, pharmacist, was a healthy man who two years ago (1904) noticed a change in the quality of his voice, to which have been recently added attacks of dyspnea and trouble in swallowing.

I saw him Feb. 13, 1906, at my clinic, and with the laryngoscope perceived a tumor of large dimensions which seemed to originate upon the left side of the larynx, covered the larynx entirely, permitting only a very little portion of the right arytenoid to be seen.

It was irregular in form, almost rectangular, and of slaty color, like a congested lung. The transverse diameter was about 4 cm. (See Fig. 5). It was movable at the free end, and in deep respiration was almost drawn into the laryngeal cavity. It was soft to the touch.

I thought of an angioma, originating from the left pyriform sinus, and my first attempts with a galvano-cautery snare causing violent spasms, I advised a tracheotomy, which was done March 5 by Prof. Sorge, at Clinique Hospital.

April 30 I commenced to destroy the tumor by the natural route, and with the aid of the galvano-cautery snare I removed large bits, then cauterized to reduce what remained of the growth. In December the destruction was finished.

Then I was able to discover that the tumor involved not only the pyriform sinus, but all the aryepiglottic fold and the false cord of the same side.

The hemorrhage after each sitting was rather severe, but by continuing the cauterizations and applications of adrenalin such improvement was gained that the tracheal canula could be removed in January, 1907.

Histologic examination confirmed the opinion that it was a cavernous angioma. (See Fig. 14.)

The rarity of such tumors, the large dimensions, the extensive seat and the excellent effect of a simple treatment in which the galvano-cautery snare played an important role, justify the report of this case in which I assisted at a progressive regression ending in complete cure.

Case 6.— A young man of 18, having a rather limited pulmonary tuberculosis,[2] was brought to my hospital clinic December 2, 1905, by Prof. Caporali. My advice was desired

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(1) Roberto Gennarelli, of Naples. No. 24 of my register A,

(2) Ricardo Brugaglia, of Marano. My register A, No. 66.

for an aphonia and an embarrassment of respiration with which he had suffered for three or four months.

Laryngoscopic Examination.—A polyp as large as a chestnut occupies the anterior two-thirds of the glottis; it is round, pale, the surface somewhat shagreened, and turns rather to the right side, although seeming to originate at the commissure. (See Fig. 6.)

December 11, with forceps of various models and under cocain anesthesia, I made several attempts at extraction, which failed. Next day, in a fit of coughing, the patient spat out the polyp entire.

Voice and breathing immediately became normal.

Upon examination the larynx appeared wholly normal, and a year later it was impossible to recognize the seat of the tumor.

Prof. Martuscelli found tubercles and giant cells upon histologic examination. (See Fig. 15.)

In 1907 the patient, who took no care of himself, succumbed to pulmonary tuberculosis.

This instance of laryngeal tuberculoma is not without interest, for, besides the contribution which it gives to the polypus or vegetating form of laryngeal tuberculosis, it shows what dimensions these tumors may attain, their relative benignity, the weakness of their point of implantation, which allows their removal with moderate force, and the difficulty of diagnosis by physical signs alone. At first I thought of tuberculoma (after the chest examination and finding the bacilli in the sputum), but the form, the size and the color of the polyp, left a suspicion of adenoma or fibroma.

Case 7.—A young woman, 26 years of age, of Catanzaro,[1] was sent to me by Prof. Scorzelli, in October, 1892, for violent suffocation attacks which necessitated her being sent to the hospital (Incurables).

One month later Prof. di Giacomo was obliged to tracheotomize her. and in December I was called to make another examination.

Laryngoscopic Examination.—A large tumor resembling in its general appearance (surface, color) an hypertrophied tonsil, the size of an almond, arising from the right half of the larynx, had invaded all the laryngeal cavity, almost touching the opposite side. (See Fig. 7.)

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[1] Rosa Marino. (See No. 45 of my private register.)

It seemed to originate from the epiglottic petiole with a large pedicle and extended to the right aryepiglottic ligament. Its mobility was moderate.

March 16, 1893, with one grasp of the forceps, I took out the entire tumor.

Prof. Oro, who volunteered to make a histologic examination, stated that it was an adeno-epithelioma, but in spite of repeated searches he was not able to find the specimen again four years later.

The patient died after a few months, of a pharyngeal recurrence.

While on the one hand this case shows us that under favorable conditions a laryngeal tumor of bad nature may be entirely removed with a good technic through the natural channel, on the other hand it proves the fatality of malignant tumors and their recurrence in regions distant from the primary seat.

Case 8.—A sailor,[1] very robust, 38 years old, whom Prof. d'Arrigo brought to me November 26, 1904, had suffered for a year with dyspnea. For two months he had received hypodermic injections of sublimate without success.

The patient had a nasal voice, had difficulty in swallowing, was very much oppressed, and the dyspnea was augmented when he tried to sleep.

Laryngoscopic Examination.—A large tumor, the size of an apricot, which, like the one in the preceding case, resembles in color and surface an hypertrophied tonsil, appears to arise from the pyriform sinus or the aryepiglottic fold on the right side. It involves all the supravestibular region in such a way that only the left arytenoid (mobile) and the internal surface of the epiglottis can be seen. It is hard to the touch and immobile.

I tried to excise at least a fragment with the galvanic snare, not having an exact idea of the tumor's size, but, as soon as the snare was well in place, such a violent spasm came on that I was obliged in haste to cut the wire.

I then advised tracheotomy, which was done at the Hospital Clinique, in December, by Prof. Sorge.

A month later, by means of a galvanic snare, I removed large portions of the tumor by the natural route, but I must

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(1) Francesco Panattieri, of the Island of Lipari. (No. 247 in my private register.)

confess that I undertook a useless task, for an enormous mass remained. I recommended sub-hyoid pharyngotomy, which was done at the hospital, in my presence, by Prof. Sorge, July 18, 1905.

After free section of the thyro-hyoid membrane we came directly upon the neoplasm. I proposed to encircle it with a platinum wire, but this was impossible. Prof. Sorge soon discovered that, as he had foreseen, the tumor was implanted all along the upper border of the right thyroid cartilage, leaving the lateral wall of the pharynx free. He immediately performed a hemi-laryngectomy.

Fig. 8, which was sent to me by my distinguished co-worker, reproduces what was removed.

Unfortunately, 21 days after the operation, the patient succumbed in collapse, probably due to pneumonia ab ingestis.

Histologic examination made after several trials by Prof. d'Arrigo, who gave me the beautiful specimen shown in Fig. 16, showed that it was an epithelioma, though the homogeneous covering gave it the appearance of an adenoma or a fibroma.

Both of these latter cases are rather interesting. Although malignant tumors easily attain considerable size, that in these two cases is remarkable. On the laryngeal side their delimination is marked, while the extra-laryngeal development is extreme.

Case 9.—An old man, 78, robust, following the profession of driver,[1] came to my free clinic in May, 1899, for a very severe dysphagia which had afflicted him for several months.

Laryngoscopic Examination.—Epiglottis flat and lowered upon the aditus. On its free border, toward the right side, there is seen a tumor as large as a nut (See Fig. 9) of somewhat rounded form, red, surface uneven, grooved by numerous varicose vessels, very mobile, going beyond the base of the tongue in forced expiration.

When respiration is quiet, or during inspiration, it falls upon the pharyngeal aditus in such a way that one can see only a part of the tumor, that which presses the epiglottis down upon the laryngeal aditus.

In the laryngeal cavity, only a little of the left arytenoid can be seen.

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(1) Giacomo Crispino, of Cardito. See No. 271, register A of my clinic, May 9, 1899.

The timbre of the voice is little modified, but the dysphagia is so marked that only liquids can be swallowed, and these even with difficulty.

Upon the backs of both hands and feet, and the external aspects of both forearms, are nodules characteristic of idiopathic hemorrhagic sarcoma of the skin, which was first manifested eight years previously.

Six days later (May 15), under cocaine anesthesia, I attempted to take out the tumor with forceps, but a lively hemorrhage came on and I had to stop. After a week of rest, with the aid of Kirstein's spatula and a snare, I was able to remove the tumor entire.

I watched the patient for three or four years and am sure that he was completely cured.

The microscopic examination by Prof. Martuscelli showed a hemo-angio-endothelioma (See Fig. 17) analogous in structure to that of a cutaneous nodule which I took for the purpose from the palm of the right hand.

Case 10.—A patient 48 years old[1] had suffered since childhood with catarrh of the upper air passages. He is gouty. His mother died at 80 of breast cancer.

For some time he had noticed rattling and defective respiration, which determined him to consult me in May, 1899.

Laryngoscopic Examination.—From the right side of the laryngeal cavity a large mass extends to the aryepiglottic fold of that side, effacing the glottis and leaving little space on the opposite side. It is irregular in shape and has a muriform appearance. The color is mainly red with silvery spots here and there. The tumor is partly mobile and appears to originate from the arytenoid cartilage to which it is adherent. Although 40 days previously the patient had spat out a fragment of the tumor as large as a bean, the dyspnea was considerable.

The patient himself drew my attention to several nodules on the left hand characteristic of hemorrhagic pigmentary sarcoma. This is why I at once suspected the real nature of the laryngeal tumor.

Eighteen days after the first visit the patient called me in haste to see him in the country, where he was having violent attacks of laryngo-stenosis. The tumor had enlarged. I prepared for a tracheotomy, but I succeeded with forceps and

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(1) Giuseppe Cardone, of Salve. No. 160 of my private practice register.

curettes in taking out, by the natural way, the greater part of the mass, which was friable and bleeding. I then introduced an O'Dwyer tube, which I left for several hours.

Ten days afterward the patient, almost recovered, came to visit me at Naples.

There remained only a small vegetation on the anterior surface of the right arytenoid, which vegetation was removed some months later with a snare by Dr. Villani, of Lecco.

In the nose were some similar growths which my colleague excised in the same way.

Prof. Martuscelli found in this case that the structure was identical with that in the preceding case; namely, hemo-angio-endothelioma. (See Fig. 18.)

These two cases seem interesting to me for the following reasons:

For a long time I have been calling attention to the coincidence of this class of tumors with cutaneous sarcomatosis. I have demonstrated their histologic analogy and their chronologic relation and especially have I brought out the enormous development which these neoplasms may reach in the larynx and isthmus of the fauces.[1]

I am satisfied that systematic examination of the skin in cases of this sort will enlarge the statistics. This is an important matter, for at the beginning Kaposi's cutaneous sarcoma may be mistaken, and although the structure of laryngeal tumors may rank them with sarcomata, often they do not recur after ablation, or, if there is a recurrence, they remain relatively benignant like the cutaneous sarcoids.

It would be difficult for me to explain why these tumors, which, ordinarily, on the skin preserve a rudimentary appearance, attain such size in the larynx. I cannot tell why sometimes we have an infiltration and sometimes a perfectly developed tumor. I confine myself to interpreting them as relatively benignant in accordance with what we know of the cutaneous form, and to saying that I have seen similar neoplasms—genuine hemo-angio-endotheliomata, after Radaeli—in the nose, behind the velum, and on the pharyngeal pillars and walls.

These ten short observations and the brief considerations drawn therefrom lead to the following conclusions:

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(1) Massei-Martuscelli-Prota: Le complicanse laringee nella sarcomatosi cutanee. Naples, 1899.

1. Certain tumors of the larynx, of good or bad nature, sometimes attain considerable volume and can be called giant tumors.

2. The exaggerated dimensions of these tumors may sometimes be the result of a transitory phase which, in spite of their benignity, may constitute a vital danger.

3. Certain hemo-angio-endotheliomata complicating cutaneous sarcomatosis reach a considerable development in the larynx.

4. Certain others, of malign nature, epitheliomata, for example, in isolated form, may attain an enormous size, especially if extra-laryngeal.

5. The large dimensions do not always permit recognizing the point of origin.

6. The physical characters of giant tumors do not always indicate their nature, so that to make a diagnosis a microscopic examination is necessary.

7. From a biologic viewpoint giant tumors of the larynx have a twofold interest: Their volume (taking account also of their implantation), which offers immediate danger; and their nature, either on account of the possibility of a regressive phase, or on account of their malignant character.

8. Local treatment by the natural route, even when a preliminary operation is indispensable, often checks the local infection and puts off the danger of a general infection, provided it be applied with exact knowledge of the neoplasm's nature.

Gentlemen, in our times, when broad culture is indispensable for a specialty, we veterans of very recently formed army note the tide which carries the young toward general surgery and tends to throw aside operations through natural routes which are more simple and frequently offer greater chance of success.

Some of the cases I have just reported were victories for surgery through natural channels, but faith is necessary to overcome the difficulties met along the way.

By the modest contribution which I am giving to this great scientific meeting, I flatter myself that I have brought to light the good that can be done for certain cases (without renouncing, for that matter, classic surgery when necessary) with a patient and delicate technic, and how much an exact diagnosis of the nature of these tumors clears up their importance and significance.

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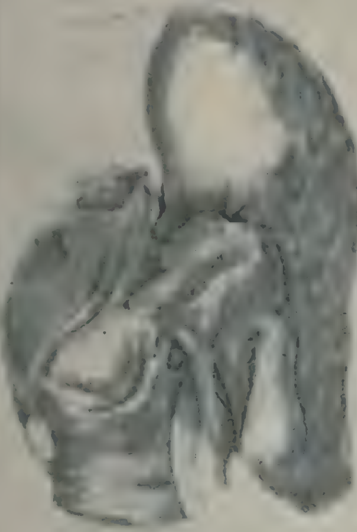

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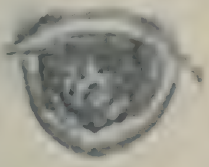

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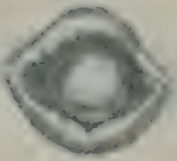

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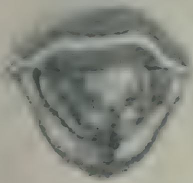

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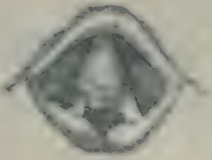

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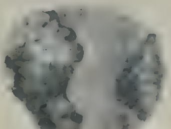

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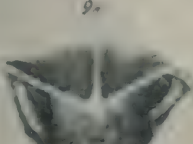

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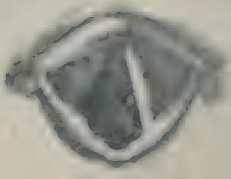

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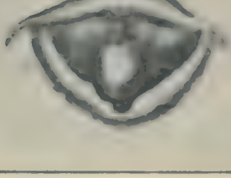

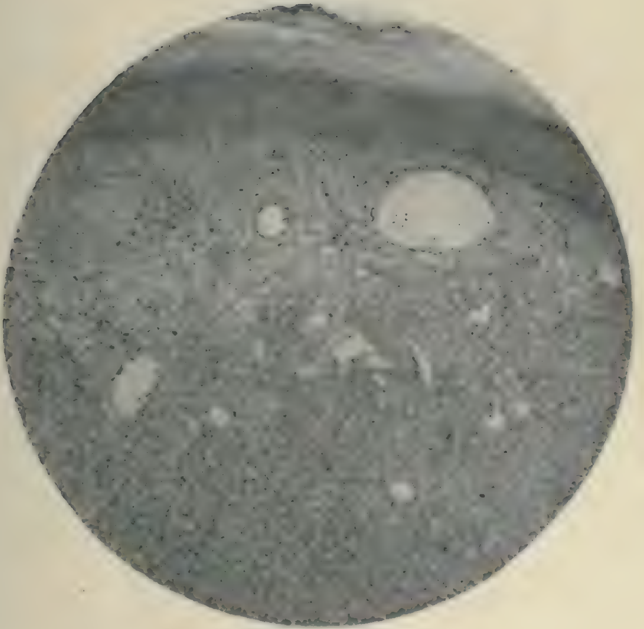

FIGURE 11.
Case 2. Cystic Fibroma. (Figure 2.)

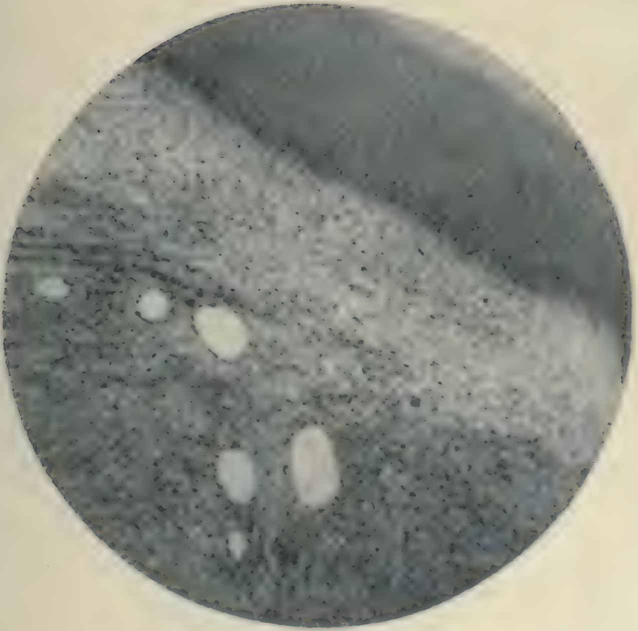

FIGURE 12.
Case 3. Cystic Fibroma. (Figure 3.)

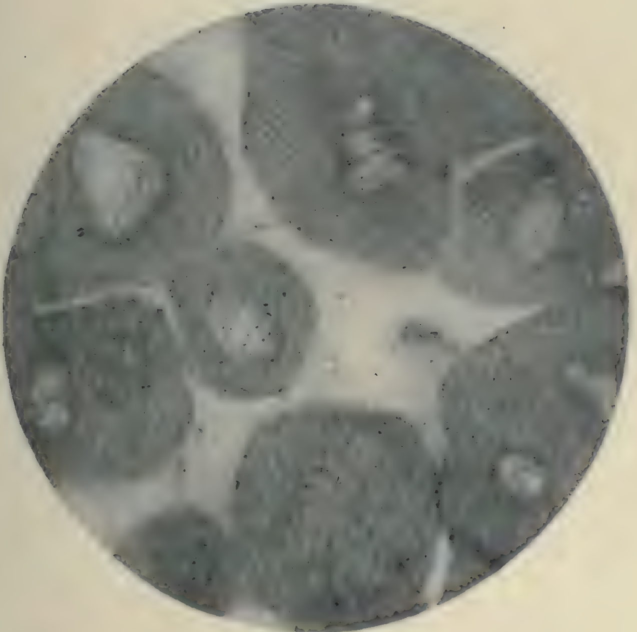

FIGURE 13.
Case 4. Papilloma. (Figure 4.)

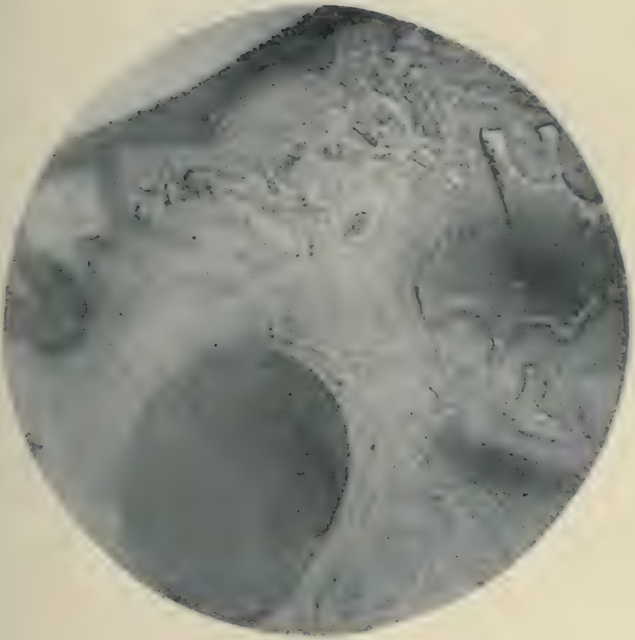

FIGURE 14.
Case 5. Angioma. (Figure 5.)

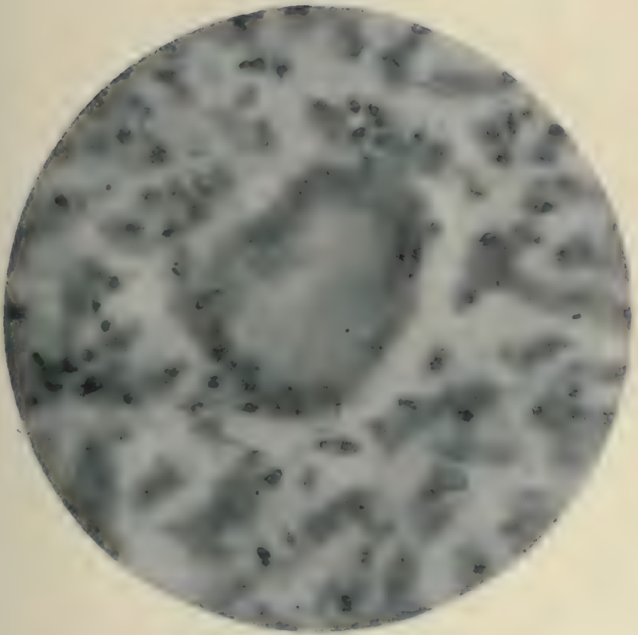

FIGURE 15.
Case 6. Tuberculoma. (Figure 6.)

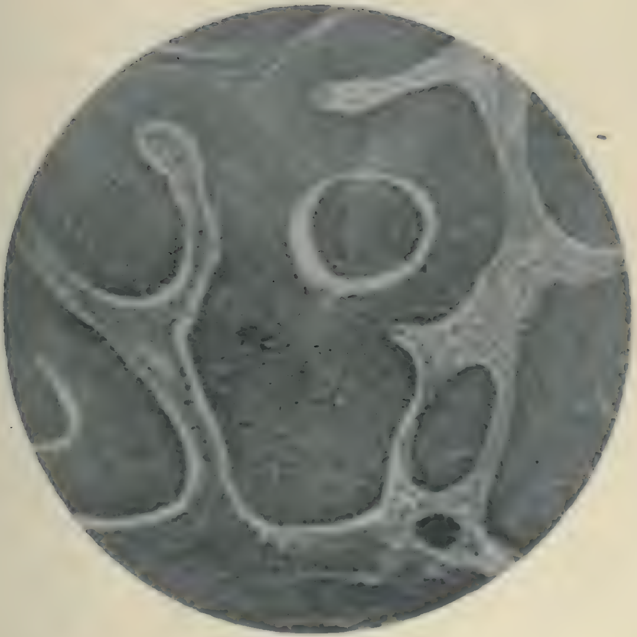

FIGURE 16.
Case 8. Epithelioma. (Figure 8.)

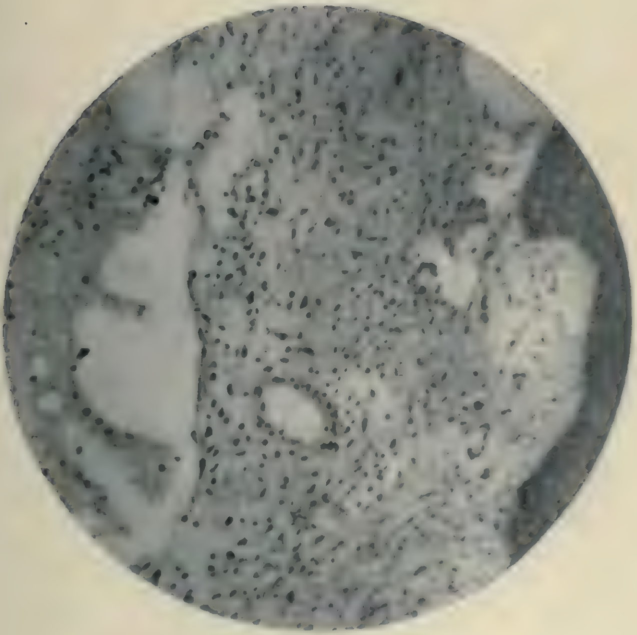

FIGURE 17.
Case 9. Hemo-angio-endothelioma. (Figure 9.)

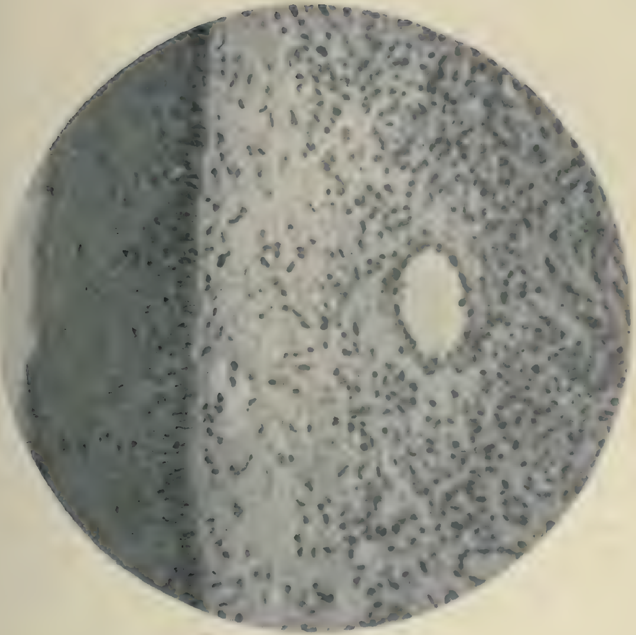

FIGURE 18.
Case 10. Hemo-angio-endothelioma. (Figure 10.)

XXXVII.

THE OPERATIVE TREATMENT OF MALIGNANT TUMORS OF THE NOSE.*

By Prof. Dr. Alfred Denker,

ERLANGEN.

The treatment of malignant growths which develop in the cavities of the body is at this time exclusively surgical. Although we have been able to influence and cause to disappear, localized tumors situated on the surface of the body by means of the Röntgen and radium rays or by fulguration, we are still unable to use these in the treatment of the malignant growths which lie in the deeper parts. In spite of the energetic efforts to discover the etiology and pathogenesis of carcinoma, which have taken place in the last few years, the attempts along this line have led to no results which allow us to hope that the near future will see a non-operative specific treatment. The different carcinoma sera and other medicinal treatments are unfortunately so far to be designated only as slight advances in the therapy of malignant tumors. Even the arsenic treatment, which has been frequently recommended for sarcomatous tumors, leaves us usually in the lurch, although it has given good results in isolated cases, especially of lymphosarcoma. Although in the malignant nasal tumors which are usually of such bad prognosis it would be very desirable to have a specific therapy, for the present we have no such method at our command, and the possibility of cure rests upon a radical operation.

In the very earliest times the attempt was made to develop an operation for this purpose, and the number of methods in use has become a very great one. At this place I will mention only those methods which completely lay bare the origin and extent of the tumor and allow its thorough extirpation. For this reason I must refrain from discussing the methods of Hippocrates, Garengot, Wutzer, Rouge, Furneaux-Jordan, and Castex, which are confined to a splitting and reflection of the cartilaginous nasal framework and do not guarantee a complete view of the field of operation.

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*Paper read before the First International Congress of Laryngology and Rhinology in Vienna, 1908.

As preliminary operations for those tumors which grow out of the deeper parts of the nose or accessory sinuses, come next in consideration those extensive operations which are also used in malignant tumors of the upper jaw, the temporary or permanent resection of the upper maxilla. Both operations are extraordinarily bloody and one is accustomed, in order to overcome the danger of aspiration-pneumonia due to the excessive hemorrhage, to make use of the preventative tracheotomy with temponade of the air passages and even the ligation of the common or external carotid, and, as of late, the intubation of Kuhn. The total resection of the upper jaw, as first performed by Gensoul in 1827, has been, in regard to the incision, modified in many ways, especially by Velpeau, v. Langenbeck, Weber and Kocher, while the division of the zygoma, the union of the upper jaw with the nose and frontal bone, and the sawing through of the hard palate, on the whole, remains the same. The mortality in total resection of the upper jaw amounts to, according to Krönlein, 21.5 per cent, and, according to König, to 30 per cent.

The v. Langenbeck osteoplastic partial resection of the upper jaw, in which the incision passes through the soft parts of the cheek, exposes the base of the skull through a temporary laying back of the upper jaw with removal of the palatal plate and the alveolar process. There are numerous modifications, of which I wish here to mention only those which take their way through the mouth; one can differentiate by the latter an intermaxillary and a palatal route.

The palatal route was first used by Nelaton, who, after splitting the soft palate and cutting through the mucoperiosteum of the hard palate in the middle line with subsequent resection of the posterior two-thirds of the hard palate, attempted to penetrate into the deep parts of the nose and nasopharynx. In like manner Gussenbauer operates, elevating the mucoperiosteal covering of the hard palate after penetration in the midline to both sides as far as the alveolar process, and then removes the palatine process and the horizontal plate of the palate bone; that is, the floor of the nose and maxillary sinus. After removal of the tumor the two flaps which have been pushed aside are brought together with sutures.

The entrance made by the Nelaton-Gussenbauer method is, according to von Bergmann's opinion, not sufficient for those tumors of the nose and naso-pharynx which come down from the base of the skull. Those oral methods which consist of a temporary resection of the alveolar process in connection with the hard palate, allow of a more free laying bare of the field of operation.

As founder of this course, we must look to Chalot, who, in the year 1886, first performed this operation. Habs, who in 1898 employed the Chalot method for the extirpation of an angio-myxofibroma, which was growing from the base of the skull, describes the method, shortly, as follows: After severance of the cul-de-sac of the upper lip with the alveolar process between the two first molar teeth, the soft parts are elevated with an elevatorium as far as the spina nasalis anterior-inferior and to the floor of both nasal cavities. Then the mucous membrane of the floor of the nasal cavities is pierced with a pointed scalpel.

After introducing two Nelaton catheters into the mouth, through the nose, an end of a Gigli saw is attached to the end of each and the two are then drawn from the nose and the septum is sawed through, from behind forward, near the floor of the nose. Then the operation proceeds on the reclining head. After supporting the lower jaw by means of a Gutsch support, the hard palate is split inferiorly on both sides from the socket of the removed cuspid-tooth along the alveolar border to the attachment of the soft palate (profuse hemorrhage). Quickly the processus alveolaris between the alveoli of the cuspid teeth is separated from the nasal cavity, right and left, whereupon the entire hard palate, which is only held by the soft palate, can easily be depressed upon the tongue. After removal of the tumor mass the hard palate is replaced and fastened by silver sutures.

The method of temporary resection of the palate published by Partsch in 1898 differs from the procedure of Chalot principally in that with the palatal plate, not only the anterior part of the alveolar process, but the entire processus alveolaris is turned downward. It is performed in the following way: Transverse incision through the mucous membrane to the fold of transmission of the upper lip; retracting upwards of the soft parts; piercing the mucous membrane of the nose at the apertura piriformis; at the same time, chiseling with broad chisels through the septum, the middle and facial walls of the maxillary antrum in a horizontal direction, over the floor of the nose and the antrum; turning back the entire hard palate with the alveolar process upon the tongue; after extirpation of the tumor, replace the palate in its normal position, and fix by suturing the mucous membrane. In 27 cases collected by Löwe and in part operated by himself after Partsch's method, the bones were united at the previous point of separation, although in part requiring a long time.

While Chalot and Partsch aim at the laying bare of the field of operation by means of the turning back of the palate, Kocher accomplishes the same end by separation of the horizontal parts of both upper maxillae by the intermaxillary path, He divides the upper lip by means of a perpendicular incision from the nasal aperture, running downwards, and then cuts through the fold of transmission of the upper lip to the bone. Corresponding to the last incision, which runs horizontal, the connection of the alveolar process and the hard palate with the facial and median walls of the antrum is cut through by means of broad chisels. If one then cuts through the hard palate and the alveolar process, in the midline, between the two superior central incisors, with a chisel or Gigli saw, both of the horizontal halves of the upper jaw may be forcibly pulled apart, and one obtains a good view of the nasal cavity and the naso-pharynx. After cleaning out the tumor mass, the union of the processi alveolares in the midline follows by means of metal suture, the hard and soft palates are then sutured, and the cleft in the lip is closed also by means of sutures. The cosmetic effect by the Kocher, as by the Chalot-Partsch, method is good; by both operations the hemorrhage, as a rule, is very great.

The nasal methods of laying bare the malignant tumors, in which, as already indicated, the operation involves not only the cartilaginous nose, but especially the bony nasal walls, can be divided, according to Kümmel, into those where the entire nose is cut and reflected, and those which require a temporary or permanent resection of the upper nasal region alone.

According to Linhart the nose is split along its posterior part, in the midline, the nasal bone and the processus nasalis of the upper jaw is detached by a chisel or a saw at the point of attachment to the frontal process, and thereupon the entire half of the nose is pulled aside without previous detachment of the skin covering, while one causes a fracture in the region of the lachrymal bone.

By the method of von Chassaignac, carefully described by v. Bruns, the skin-periosteal incision is made from the inner canthus of the eye, in a circular direction over the ridge of the nose and its attachment, back to the ala nasi of the first side. The bony parts of the nose and the septum are cut through with a saw or chisel and then the entire nose is turned back to the opposite side.

According to the method recommended by Ollier, the nose remains attached to its lower line of insertion. The section runs on both sides from the root of the nose along the attachment of the outer nose to the line of attachment of the alae nasi. Section of the nasal bone, the processi nasales of the superior maxilla and of the septum in the plane of this incision by means of the saw, whereupon the nose usually may be turned downward without severing the cartilaginous portion. The cosmetic result in this operation is said to be not always very favorable.

Methods have been described by v. Langenbeck, G. Killian, A. Barth, Gussenbauer, Küster, Winckler and more recently by Moure and Brockaert which aim to lay bare the upper and posterior portions of the nose by means of a temporary or permanent resection of the nasal bone and the processus nasalis of the upper jaw. v. Langenbeck* made an incision from the middle of the glabella to the end of the nasal bones and from there along the apertura piriformis to the alae nasi. The skin flap thus outlined is dissected back, care being taken not to injure the periosteum; then the nasal bones are separated from each other near the septum, and the base of the processus nasalis is cut through with a saw deep into the orbit, from within outward. The os nasale and the processus nasalis of the superior maxilla, which remain joined to the mucous membrane and periosteum can be broken outward and later replaced.

G. Killian (and similarly A. Barth) makes a cutaneous incision in the median line from the glabella to the middle of the nasal ridge; after the periosteum has been retracted in the frontal region, the frontal sinus is opened, a probe inserted into its duct, and this is followed by detachment of the nasal bone from the frontal bone without disturbing the skin in this region. The os nasale and the skin are turned outward and, finally, the portion of the nasal process which still remains is chiseled away from the frontal bone as far as the probe. Winckler goes still further to the side and turns the mucous membrane-periosteo-bone flap downward.

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*See Kümmel, Handbuch der praktischen Chirurgie, by v. Bergmann, v. Bruns and v. Mikulicz. Vol. I. p. 833.

Inasmuch as the access to the ethmoid cells by this operation is a very small one, G. Killian, especially in connection with the radical operation on the frontal sinus, anticipates the laying bare of the whole ethmoid labyrinth with the permanent resection not only of the nasal bone, but also most of the processus nasalis of the superior maxilla.

According to the method of Gussenbauer, who lays bare the upper nasal segment on both sides, the incision runs from the middle of one eyebrow, along the orbital border, to the proc. nasal. max. sup., then straight across the nose, corresponding to the borders of the nasal bones, and finally on the other side, upwards in the same way. Both proc. nasal. max. sup. are cut through with a sharp chisel horizontally as far as the margo orbitalis, then frontally in continuity with the lachrymal bones, the lamina papyracea, the proc. nasal. frontalis, and finally the lamina perpendicularis ethmoidalis to their junction with the vomer. The whole flap of soft and bony tissues may now be turned upward. If, as Winckler proposes, a perpendicular incision is made on both sides at the ends of the incisions in the eyebrows, the anterior walls of both frontal sinuses may be temporarily resected in one piece, together with the above flap, and these sinuses opened at the same time.

According to the method of Küster, which is a modification of Chassaignac-v. Brun's operation, the cartilaginous nose is separated from the bony by a cross incision, the former is left in situ, the latter detached according to the Chassaignac-v. Brun method and turned to the side.

In the method published by Moure in 1902, a skin incision is made from the medial end of the eyebrow downward to the nasal opening, ending in the upper lip at the height of the insertion of the ala nasi. The soft parts are detached from the nasal bone and the nasal process of the upper jaw as far as the lachrymal bone, care being taken to prevent an injury to the cutaneous part of the ductus nasolacrimalis which would result in a fistula. A part of the nasal process of the upper jaw, a part of the lachrymal bone, the nasal bone, the spina nasalis and the spina frontalis are resected. In this way a large access to the ethmoid is obtained, and it is possible to remove this in toto. After resection of the ethmoid, diseases in the region of the sphenoid can be attacked. After primary closure of the wound, only a slight depression at the inner canthus remains. (Abst. in *Internat. Centralbl. f. Laryngol.*, Vol. XIX, p. 327.)

In 1905, Brockaert extended Moure's method to the sphenoid cavity by detaching the periosteum from the facial wall of the upper jaw and reflected it with the soft part upward. After resection of the facial and medial walls of the sphenoid and the inner quarter of the orbital border, a tumor, a sphenoid endothelioma, could be easily removed. Union of the lips of the wound eight days later. (*La Presse Otolaryngol., Belge*, No. 3, 1905.)

Naturally, I have not been able to describe all the numerous combinations and modifications, but only the principal methods which are used in extirpating malignant nasal tumors. All the above methods, with the exception of the Chalot-Partsch, have in common that they cause a lesion of the continuity of skin of the face and result in a scar or a greater or less cosmetic defect. Most of them are very *bloody*, some, especially the Chalot-Partsch and the Kocher, are accompanied by an enormous hemorrhage, which, according to Kümmel, make a prophylactic tracheotomy and the introduction of a tampon-canula very advisable.

About two years ago, as most of my German colleagues are aware, I published a method which obviates the cutting of the skin of the face and materially lessens the hemorrhage, while still allowing the field of operation to be made completely visible. The method was an *oral* one, in which, after the bone had been laid bare subperiosteally, the whole facial and medial wall of the antrum as well as a part of the nasal bone and of the proc. nasalis of the superior maxilla were permanently resected. For the benefit of my non-German colleagues, I will here describe the operation in detail. It is based on the operation for old, chronic empyema of the antrum, which was described in the *Archiv. f. Laryngol.*, 1905, Vol. XVII, part 2, and is performed as follows:

Unless the administration of ether is contraindicated for other reasons, we use the morphine-ether narcosis (dose of morphine for adult usually 0.014). In addition, to reduce the hemorrhage, a cocain-adrenalin tampon, in the form of strips of gauze, shoved as far back and up as possible into the nasal cavity, is recommended. Several strips of gauze leading out of the mouth are placed between the last teeth of the side to be operated, and the upper lip is drawn upwards and outwards by two blunt hooks. An incision is made through mucous membrane and periosteum down to the bone, which begins above the wisdom tooth, in the fold of transmission of the upper lip, runs forward, horizontal to the alveolar process, cuts through the frenulum labii superioris and extends 2-3 cm. further in the same direction over onto the other side. The soft parts are pushed upwards with the raspatorium and held by two four-pronged hooks until not only the superficies facialis maxillae superioris and the region of the apertura piriformis, but also the lower part of the nasal bone and the proc. nasal. max. sup. near the margin of the lower and inner orbital border are laid bare. Only when this has been thoroughly accomplished can a sufficient access to the ethmoid be obtained. This strong upward retraction of the soft parts occasionally tears the mucosa of the meatus at the apertura piriformis. The nervus infraorbitalis is drawn upward with the rest of the soft parts.

If by previous rhinoscopy it has been found that the tumor has not destroyed the medial antrum wall and grown into the antrum, before the antrum is opened, the mucosa, starting at the lateral margin of the apertura piriformis, is stripped from the lateral wall of the lower and middle meatus by means of a flat elevator which is about 6 mm. broad and has an end curved at an obtuse angle. This stripping, which must be done very carefully, so that no hemorrhage into the nasal cavity is caused by lesion of the mucous membrane, extends in adults from the apertura piriformis 4-5 cm. backwards to the posterior antral wall. If the lower turbinate still exists, it is cut off at the crista turbinalis with a pair of strong nasal scissors and a piece of gauze is inserted between the detached mucosa and the bone, in order to check the moderate hemorrhage.

During the opening of the antrum from the canine fossa, which now follows, it is important not to remove the subjacent mucosa with the bone, but, while taking care not to injure the former, to remove a piece of bone at least the size of a 10 pfennig piece by means of superficial chiseling and slender bone forceps A premature hemorrhage into the antrum or nose is thus prevented. If the presenting and usually thickened mucosa is then excised, the resulting hemorrhage can easily be controlled by quick introduction of tampons. After the facial wall has been completely resected, the mucosa of the entire antrum with all of its recesses is carefully removed. This is necessary, since in most cases the antrum is involved in the process either by an extension of the tumor from the nose or by a chronic, purulent inflammation of the mucosa.

Next, the nasal wall of the antrum, with all of its processes, as far as the apertura piriformis, is completely removed with Luer's forceps and chiseling as far as the posterior wall of the cavity, so that the antrum is separated from the meatus only by the mucosa of the lateral nasal wall. At the same time, the lower part of the nasal bone and of proces. nasalis max. super. is resected in the neighborhood of the lachrymal bone.

So far, and this seems to me of the greatest importance, the entrance of large amounts of blood into the nasal cavity and from there through the choana into the remainder of the air passages has been prevented. If it is desired to prevent the downward flow of blood during the subsequent removal of the tumor, this can be accomplished by the introduction of a firm tampon into the naso-pharynx. Usually, however, at this point the patient is no longer in deep narcosis, so that we can with certainty count upon the presence of reflex swallowing of the blood and no longer need fear its aspiration. From now on, it is desirable to operate as quickly as possible. If the mucosa at the apertura piriformis has not been ruptured during the upward reflection of the soft parts, a puncture is made in the lateral nasal wall, at its anterior end, near the floor, a blunt-pointed knife of pair of scissors is introduced through the opening, and the whole wall is quickly excised and removed in toto. If the tumor springs from the middle meatus or the lower turbinal, it is removed for the most part with the mucosa. After the removal of the medial antral wall, the entire interior of the nose is broadly exposed, and it is an easy matter to cleanse the ethmoid thoroughly with conchotom and curette, and to lay bare the anterior wall of the sphenoid cavity completely and resect it. For this part of the operation, the use of artificial illumination by means of the head-light or reflector is very desirable. A view is obtained of the naso-pharynx through the choana, and tumors of the pharyngeal vault can easily be attacked and thoroughly removed, especially if the posterior part of the septum is resected or pushed to one side.

The survey of the entire field of operation allows nothing further to be desired, as even entrance to the frontal sinus is easy after the anterior ethmoid cells have been removed. In the cases of extension of the malignant process to the sinus frontalis, it is urgently recommended that the anterior wall of this cavity be resected—incision through the eyebrow and retraction upwards of the soft part—in order to remove all diseased tissue.

After careful removal of all suspected soft and bony parts—whereby care in the region of the lamina cribrosa and lamina papyracea is demanded in order not to injure the dura or the periosteum of the orbit—the wound is tamponed with iodoform gauze and the oral mucosa sewed. Applications of Burrow's solution or boric acid solution are made to the operated side, and lavage of the mouth with boric acid solution. After 3-4 days, the tampons are removed through the meatus, and on the 4th or 5th day, the stitches are removed. After removal of the tampons, dermatol and, later, boric acid are insufflated. From the 10th day on, lavage with boric acid solution by the patient, by means of a wide, curved glass tube, until the secretion ceases. Covering of the wound is accomplished by granulations springing up from the bare bone, which are gradually epidermized from the nose. It should be noticed here, that permanent disturbances of the lachrymal apparatus, caused by resection of the greater portion of the ductus naso-lachrymalis, which occurs during removal of the lateral nasal wall, has never been observed.

Furthermore, loss of teeth in consequence of severing their nerves and vessels, never happens. However, it is better not to remove the facial antral wall up to the very bottom of the alveolus.

Altogether, I have used this method seven times for extirpation of tumors and twice have used it with very good results for the radical operation of very severe simultaneous empyema of the antrum, ethmoid and sphenoid.

As to the results of the seven operations for tumors, I may be allowed to report briefly here: The first five cases have already been published in the *Muensch. med. Woch.* (1906—No. 20), and in the *Archiv. für Laryng.* (Vol. XIX, part 3). Of these, Case 2 of my first publication is excluded from our discussion because it was a case of a nasopharyngeal fibroma arising from the fissura pterygopalatina for whose extirpation our operation is not suitable.

In Case 1 (Antone R., age 23), small-cell sarcoma, arising in the ethmoid labyrinth, there appeared, as already stated, a small recurrence after two months, which, since it hung down from the region of the nasopharynx, was removed with a galvano-cautery snare. The patient, who refused further treatment, and put himself in the hands of quacks, died about a year later, of the disease.

The second patient (John D., age 41), large epithelial carcinoma, which sprang from the middle meatus, and had destroyed not only the upper portion of the medial antral wall and a portion of the antral roof, but also the greater portion of the lamina papyracea of the ethmoid. I was able to present this patient a year and a half after the operation in my course for physicians, entirely free from recurrence and without any symptoms. Three months later, however, he returned with a recurrence located on the floor of the nose, which was removed after reopening of the oral opening. The course so far (3 months after second operation) has been favorable.

In the third case (Mrs. Marie D., age 65) there was an extensive endothelioma which had destroyed the facial and medial walls of the antrum and completely filled the nose and antrum, bulging the cheek forward. Furthermore, the bone of the alveolar process was already destroyed in its posterior half as well as that of the adjacent floor of the antrum. The tumor originated in the middle ethmoid cells. The case resulted in normal healing of the operation wound, and so far, one and three-quarter years later, there has been no recurrence.

Case 4 (Mrs. Emma Z., age 73), large, medullary carcinoma, which originated in the most posterior ethmoid cells and extended into all the accessory cavities and through the lamina cribrosa into the cranial cavity. Three days after the operation, she died of meningitis following injury to the dura during the removal of the tumor from the floor of the anterior cranial fossa.

Case 5 (Joseph F., age 32), spindle-cell sarcoma, which sprang from the lamina interna of the processus pterygoideus, penetrated into the ethmoid and sphenoid cells and filled the left nasal cavity from its floor to the lamina cribrosa of the ethmoid. Two months later there was a recurrence, which was operated five months after the first operation. In spite of thorough exenteration of the cavities, with removal of almost the entire lower and medial wall of the orbit, tumor masses soon reappeared. Treatment with arsenic and Roentgen rays had no effect. As a radical removal of the tumor was no longer considered possible, treatment was limited to palliative measures, such as the cautery snare and point.

Case 6 (Lina St., age 8), fibrosarcoma arising from the lamina cribrosa of the ethmoid, pushing the septum strongly to the other side and entirely filling the left nasal cavity. Here, too, the tumor recurred in two months. At the second operation, the medial and lower walls of the orbit, with the exception of a spur on the lower border of the orbit, were resected and the greater part of the posterior antral wall and the posterior part of the floor of the antrum were removed. Nevertheless, after scarcely two months, tumor masses again came down from the nasal roof. Strangely enough, the histologic examination of the tumor masses extirpated at the second operation and that of the *second recurrence* showed a change in the character of the tumor. Whereas the earlier examinations had resulted in the diagnosis of fibrosarcoma, the later ones gave the picture of a fibroma, in places rich in cells.

If the results of the above operation are examined as to their permanency, it will be seen that they are not especially brilliant. Only one of the six cases remains thus far (1¾ years) free from recurrence. Furthermore, in cases 2 and 6, the course after the second operation allows the possibility of a permanent cure. Also, it must be noted that in all of our patients the cases were far advanced and by their extent, their histologic characters, and their origins, made the prognosis very unfavorable. I may be permitted in this connection to cite Kümmel, who wrote on malignant tumors of the nose in the Handbuch f. Laryngologie and in the Handbuch du praktischen Chirurgie. He says: "The prognosis of sarcoma is absolutely dependent upon whether or not the tumor can be radically removed. If that is impossible, the prognosis is entirely bad. If it is possible, the prognosis and results vary widely, according to the location, age, and histologic character of the tumor. The worst prognosis is in the case of *round-cell* sarcoma, especially the small-cell variety (Case 1), which are the most suspicious and recur with greatest frequency. The few cases in the literature which have been followed for a long time ended almost invariably in death, although it was frequently possible to give the patient relief for a long time by repeated operations. The spindle-cell sarcomata and the fibromata are as a rule less malignant, yet the writer had inoperable recurrences of these also" (Cases 5 and 6).

"In tumors arising from the roof of the nasal cavity, the prognosis is very grave on account of the danger of entrance into the cranial cavity" (Case 6).

"The tumors which, from any source whatever, have penetrated into the ethmoid can scarcely ever be removed by a radical enough operation." This was the case in all of our patients.

"The prognosis of nasal carcinoma (2 and 4) is much worse than of sarcoma. Most cases in literature have, it is true, been operated, yet not one seems to have had a definitely favorable outcome, although in certain ones death has been postponed for a long time (3¾ years, Schmiegelow)."

From these words of Kümmel it is evident that the prognosis of malignant nasal tumors, even with the very best of preliminary operations, is a very poor one. I do not need to state that I do not expect any great improvement of the prognosis by my method of operation. I do believe, however, that in this way there can be expected a diminution of the mortality from the operation itself, independent of the other advantages, which will be noted at the end of the article. First, however, I would like to discuss the articles in regard to the treatment of nasal tumors which have appeared since my publication of two years ago.

Two cases of endothelioma were operated according to our method, in 1907, in the Strassburg Ear Clinic, and have been described by Althoff in the *Archiv. f. Laryng.*, Vol. XIX, part 2. The director of the clinic, Prof. Manasse, told me that my method had given him very good service, that the view of the antrum, ethmoid labyrinth and sphenoid sinus had been a very good one, and that in this way these cavities could be very well cleaned out.

Prof. Neumayer, Munich, operated an ethmoidal sarcoma, a carcinoma and a severe tuberculosis of the nasal meatus by our method and was *very well satisfied by the view which this method gave of the origin and extent of the disease processes.*

In June, 1907, Delie reported in the Jahresversammlung der belgischen oto-rhino-laryngologschen Gesellschaft a case of carcinoma of the lower turbinal which he operated in almost exactly the way I have described, with the best results.

In the *Monatschrift f. Ohrenheilk.,* 1907, Vol. IX, Schoetz reported a case of carcinoma of the antrum, from Kümmel's clinic in Heidelberg, where our method of operation was followed. The patient could be discharged for ambulant treatment 10 days after the operation. Schoetz states that "the good cosmetic results and the quick cure would make this method the typical operation in the future."

In the second part of his work, "Zur Chirurgie der Nase," published last year, Löwe published a method of laying bare the ethmo-sphenoid region from the mouth, to which he came independently of me. This method almost completely corresponds with mine, differing only by the fact that Löwe, in order to uncover the cells lying in front of the ductus nasofrontalis, temporarily reflects the lower part of the nasal bone upwards, whereas I resect it. That the permanent removal of the lower third of the os nasi causes no deformity may be seen from this photograph.

In an article which has recently appeared in the *Archiv. für Laryngologie,* Uffenorde, while describing the methods of operating on the accessory cavities, stated that my method had great advantages in tumors of the antrum and tuberculosis of the medial wall, whereas in affections of the ethmoid, access to the anterior ethmoid cells was very difficult, and even impossible. This, however, is the case only when the soft parts have not been retracted upwards sufficiently, and the lower part of the nasal bone and the proc. nasal. max. super. have not been removed. I admit that I did not emphasize this part of my operation in my first publication, and am convinced that

Uffenorde would modify his opinion as to the view of the field if he resected these bony parts, in suitable cases.

To you, however, who are present, I hope to convince directly by demonstrating the view of the field of operation on a specimen which shows my method. (Demonstration.)

In conclusion, I desire to summarize the advantages of my method over the extensive preliminary operations used heretofore.

I. My method allows as well as any nasal, facial or oral method a wide and accessible laying bare of the origin and extent of tumors in the nose and accessory sinuses. Accordingly, it fulfills the condition of permitting the thorough exenteration of the tumor.

II. There is no cutting of the external skin, such as is found in all nasal and facial methods. Since the removal of the bony portion causes no deformity, the cosmetic result is an ideal one.

III. The downward flow of large quantities of blood is almost certainly prevented by our method. This makes it preferable to the very bloody operations of Chalot, Partsch and Kocher, which, on the other hand, have very good cosmetic results. As a result of the lessened danger of aspiration pneumonia, our method obviates the prophylactic ligation of the carotid and the preliminary tracheotomy. The mortality from the operation itself is very small.

IV. The after-treatment is exceedingly simple, the healing usually normal. As a rule, the patient can be discharged from hospital treatment in 10-14 days after the operation.

For these reasons, I think I am justified in warmly recommending my method for nasal tumors, and requesting you to test it.

XXXVIII.

CYSTS, ABSCESSES, AND EDEMA OF THE EPIGLOTTIS.*

By Henry L. Swain, M. D.,

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NEW HAVEN.

Swelling of the epiglottis, occurring suddenly and independently of any of the more chronic and systemic conditions, such as syphilis and tuberculosis, is apparently due to a number of causes, and three peculiar instances having come to my notice with this condition at about the same time, I was led to give the matter enough thought to look over my own records and to find out the opinions of others as to the origin and causation of such cases. The results of this consideration I venture to present to you under the above title.

Two patients with enlargement of the epiglottis came to me, in whom there was a history of previous attacks similar to the one in which I first saw the patient, and each of the attacks was apparently not connected with any previous inflammation of the tonsils or other parts of the throat.

In 1895 I was consulted by Mr. L. C., 46 years old. He had had difficulty in swallowing for at least two years. At all times there was a sense of fullness in the throat. Frequently the difficulty in swallowing solids was very great. Rarely until the present time had he had any actual pain. He had suffered in the past from frequent attacks of tonsillitis, which have presented no other difficulties than is usually the case with tonsillar inflammations. Examination showed him to have simple enlargement of both faucial tonsils. In the lower part of the throat the laryngoscope discovered a large swelling, a little to the right of the median line, on the lingual surface of the epiglottis, pressing it so far backward toward the posterior pharyngeal wall as to completely conceal any view of the interior of the larynx. The surface of the swelling was red, and it was evident from appearances that we had to do with a cyst, which was in a state of inflammation. Under simple local anesthesia a large slit was made in the wall of the cyst and a very thick, gelatinous, greenish colored fluid was evacuated. The sac seemed to be fairly thick-walled, but on seeing the patient five days later the cyst had, however, so contracted that it was impossible to get into it again. My idea had been to cauterize the lining membrane of the sac.

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*Read at the thirtieth meeting of the American Laryngological Association held in Montreal, May 11 to 13, 1908.

Nothing more was heard of the patient until January, 1896, some three months later. The sac had filled up again, to a small extent. Puncture was made, and a bead of nitrate of silver on a probe was passed down through the opening into the interior of the sac.

In October, 1898, the patient again presented himself with a large swelling in the same location, this time weighing down the epiglottis to such an extent that there was considerable difficulty in breathing, and severe choking when liquids were swallowed in large amount. Considerably more pain and constitutional symptoms were also present at this time than in the previous attacks. A liberal slit was made rapidly into the superior wall of the sac, and a large amount of mucus, with more pus in it than before, was evacuated. The collapse of the sac was so complete, owing to the gagging of the patient, that it was impossible for me to get into the opening again with a probe in order to cauterize the interior. Examination a month later showed no evidence of any cyst wall remaining.

The patient reports at the present writing that he has had no trouble from a recurrence of any inflammation in the sac, and he attributes his immunity for these years to the fact that he had his tonsils cauterized. As none of his attacks of enlargement of the cyst have followed tonsillitis, directly or indirectly, I do not take his suggestion seriously as regards the reason of his immunity.

The second case of abscess formation in the epiglottis, of the recurrent type, and of cystic origin, occurred in a young man, about 26 years of age, who was first seen by me in 1894. He was then recovering from what he now supposes to have been abscess in the region of his epiglottis. His history at the time of his first appearance was that he had had "an awfully sore throat," which he considered to be quinsy, that it had broken in the night, and, that as he had had two previous attacks of it, one in 1890 and one in 1892, he wished me to advise him as to some method of relieving him of such recurrences in the future. He was then a student in the University, and I saw him a number of times, but as his tonsils were not very large, did nothing for him further than the ordinary treatment for the catarrhal condition which existed in his nose and throat. At that time the existence of a cyst in his epiglottic region was not known to us. He had an interval of immunity of about eight years, during which time he had so much cough and bronchial trouble that he thought that he was about to become a victim of tuberculosis, and passed his time in a warmer climate, being principally engaged, in one way or another, in business in the southern states.

In 1904 he came to me with exactly similar symptoms and conditions as depicted in case 1. A large cyst or abscess cavity was found at the base of the epiglottis a little to the left of the center, which drove the epiglottis downwards and backwards over the entrance to the larynx. He was in the greatest possible distress when he came to me, having been in this condition for three or four days, and having sought relief at the hands of various physicians, all of whom, however, had failed to use the laryngoscope. Immediate relief was given by puncture and evacuation of the cyst, and the same glairy, thick, yellow mucus in large mass, mixed with small flocculi of pus was evacuated, as in case 1. There were so much blood and discharge, that it was impossible to do anything toward further opening of the sac or cauterizing its interior that night. The patient failed to come back until the third day, when the epiglottis had so righted itself and the parts had so shrunken, that it was impossible to get a probe into the opening as one would have expected, had there existed a large cyst cavity.

His description of his feelings and symptoms in the three previous attacks left but little room to doubt that he had had a similar state of affairs, with spontaneous rupture of the sac.

A three year interval elapsed, and in August of last year he again had a sudden attack of violent pain and swelling and choking, following the use of his voice in a lecture. He had had absolutely no previous inflammation in his throat nor any cold. He was then in a distant part of the country, and the sac was opened by a skilled hand, and an attempt was made to curette the interior of the sac, which resulted in an enormous amount of swelling and discomfort, and the thickening went down very slowly, not having entirely subsided a month thereafter.

He had another attack in December last, coming on in exactly the same way as before, and it was again opened, this time, however, by simple incision. The sac collasped completely, and since that time it is impossible to see any evidence other than that of the scar, which is situated at the base of the epiglottis, entirely off from the base of the tongue.

He reports that his mother and two sisters have had abscesses in their throats, which on two different occasions have been diagnosticated as being somewhere at the root of the tongue. None of these patients was ever examined with the laryngoscope.

These two cases, it seems to me, are to be diagnosticated carefully from those of abscess of the lingual tonsil, which cases of lingual quinsy are like the faucial type in that they are more or less peritonsillar and do not as a rule involve the epiglottis at all, except as the latter becomes edematous. Of the abscesses in this region which I have seen, and which number only five, three were on the left side of the base ot the tongue, just at the entrance of the pyriform sinus. At least that is the region in which the greatest swelling occurred and where I made the incision to evacuate the contents. This means, of course, just beyond the lymphoid tissue of that region. The other two cases were nearly central, a little to the right, in the fossae glossoepiglotticae, at the root of the tongue. These five cases were all subsequent to well developed acute follicular inflammation of the lingual tonsil. The two cystic cases, first mentioned, were so situated as to be clearly of epiglottic origin. The fact of the recurrences and the character of the contents in both cases, not being simply pus, but thick, glairy, greenish mucus, have led me to the conclusion that they must be cysts of the branchial cleft type. I am more impressed that this is the explanation, because of the character of the matter contained in the cysts which was exactly similar to that which I once saw in a recurring cyst which opened externally in the neck in the thyrohyoid space.

This was a most interesting case of this type, and was where we could study it more carefully than when occurring within the throat. It was in a girl of about 20 years. When I first saw the patient it had been opened frequently, evacuated, and washed out with various solutions, all to no effect, for in a shorter or longer time, the secreting mucous membrane not being destroyed, it would again fill up. When I opened and carefully probed the cavity, it went down to within so short a distance of the interior of the larynx that the separating membrane was apparently as thin as thin note paper. That this was the case was definitely demonstrated when a surgeon, rather against the advice of the laryngologist, attempted to dissect the sac out under general anesthesia. Notwithstanding that the membrane was well defined, it was impossible to loosen and remove it over a circular area of about 3/16 of an inch in diameter because of the fear of producing a fistula by opening into the interior of the larynx at the base of the epiglottis. The unremoved area of mucous membrane was lightly touched with a pure stick of silver nitrate, and the edges of the wound sewed up. For some years there was no return, and the case has passed entirely from my knowledge.

As is well known to all, in the depths of the branchial clefts of the embryo, the epiblast and hypoblast, as seen in serial section under the microscope, are separated by the merest network of a single layer of intervening fibres. Whether the inclusion cyst comes on the one side of this partition or on the other is probably purely accidental, but when, after the habit of these cysts, after years of quiescence something starts them to secretion, in the one instance it will be external and in the other at the base of the epiglottis. The clinical phenomenon is the same in both cases. Dermoid cysts and supernumerary thyroid glands are also recorded as occurring in the region which we have under consideration, so that our cases of branchial cysts fit in with the universal experience. How small these cysts may have been in the quiescent period one does not know. When inflammation starts, as in the two cases which I report, the sac becomes as large as a small English walnut. It should be remembered that on several occasions in both cases the attack came on suddenly and a large amount of pus was evacuated, making it appear that the infection had taken place in an already large cystic cavity. In these instances there seemed to have been no previous inflammation in the throat, at least none of which the patient was conscious until the feeling of discomfort resulting from the swelling of the cyst and the distortion of the epiglottis came to pass.

The foregoing explanation for these cases seems to be fairly clear, and the indications for treatment are evident and plain, the only trouble being in the difficulty of destroying the lining membrane of the sac at the time of the first incision.

During the last year three patients with simple, what might be called, "idiopathic," edema of the epiglottis came to my attention, which also were not connected with previous inflammatory action in the tonsils or throat. Two ended with distinct abscess of the epiglottis, and the third, between a morning and an evening visit was so much improved, during a nap which the patient had taken in the middle of the afternoon, as to lead one to the only probable supposition that perhaps also here a small abscess had broken. The histories are very briefly as follows:

A student of the University came in to me one evening complaining of a swollen and tight feeling in the lower part of his throat, with difficulty in swallowing. It had come on during the last two days, being very much worse today. He had had no previous sore throat or cold. Examination of his throat showed the left side of his pharynx somewhat congested, no active tonsillitis in evidence, but an enormously swollen epiglottis with a greater prominence on the right half, on its lingual side. As there was no marked swelling of the aryepiglottic fold or the arytenoid cartilages, the condition did not seem to me to be urgent and I did not make any punctures, simply spraying the throat and ordering a steam atomizer, with Seiler tablet solution, and adrenalin every two hours through the night. He came next day with a distinct white point on the more swollen right side of his epiglottis, which on puncture developed a cavity as large as an orange seed. This evacuated itself, with complete collapse of the walls. The swelling promptly went down in two or three days, and he made an uninterrupted recovery.

This case was scarcely off my hands when I was called to treat an elderly woman whom I had known professionally for a good many years. I had seen her throat at various times, and had been particularly conversant with the base of the tongue, for she had had enlargement of the lingual tonsil which frequently produced cough. She sent for me one day with the statement that she had a sore throat in a different position than ever before, and that it was exceedingly distressing to her, and that she had a queer suffocating feeling that was very disturbing. Examination showed an epiglottis very similar to the one I had seen a few days before with the exception that there was more swelling on the left side than on the right, and that the aryepiglottic fold around to the arytenoid was very considerably swollen. Here the indications were certainly for more active treatment, and paying but little attention to the epiglottis, I punctured the aryepiglottic fold, and was gratified to find that in a few hours the swelling went down in response to the puncture and the steam atomizer, used as in the other case. The next day a white point was seen upon the most swollen portion of the epiglottis, and on the following day it was punctured, with the evacuation of a considerable amount of pus, with a quick subsidence of the symptoms. In this instance I was able to put a pledget of cotton dipped in strong carbolic acid directly into the cavity itself. There had been to my certain knowledge in this patient, as I had seen her within a few days of the time she called me to the house, no active inflammation in the throat such as one would usually expect to find in an edema case.

At a time not very distant from these two cases, I was called suddenly in the night to a patient who, being a physician, diagnosticated his own case. He said he believed he was having edema of the glottis. He had been having leptothrix of both tonsils, and had had considerable irritation in this region from the foreign organism. During the twenty-four hours previous to his calling me his throat had been a little sorer than usual and a different sort of soreness than he had been having during any of the time. I found a well-swollen epiglottis, with the aryepiglottic fold on the left side also markedly swollen back on the arytenoid itself. The swelling was of the very baggy type on the epiglottis, but not so deeper down, so that I felt no alarm that the patient would immediately be troubled for breath, but nevertheless, I made multipuncture and started the steam atomizer. The patient was very uncomfortable and had a good deal of pain and trouble during that entire day. The next morning the swelling had gone over decidely on to the other aryepiglottic fold. No evidence of any pointing as of abscess in the epiglottis was seen although it remained distinctly swollen. From the morning of the second day the patient began to take moderate doses of salicylic acid and oil of wintergreen. He had a nap in the afternoon, from which he awoke very much relieved, and to my great surprise, when I examined him, the swelling had disappeared by more than half in all the parts that were affected, and I have felt fairly positive that there must have been some small abscess which had ruptured, which had been so situated that I was unable to see it.

The explanation of the first two cases in this group was rather difficult. The third and last may be said to be due to the leptothrix of the tonsils or to the gout, owing to the sudden improvement after taking the salicylates. The simplest explanation to give of the phenomena in the first two cases is that we had perichondritis with edema and the formation of a focus of pus. In both, the pus formation was spontaneous and not the result of any infected wound of the particular region where the abscess occurred, for no punctures were made in that area.

Concerning the probability of such a perichondritis, I was interested to run through such literature as I happened to have at hand, with the following results: D. Braden Kyle speaks of an acute epiglottiditis as occurring, and that it might sometimes be of a malarial or miasmatic origin. This last was referred to by several other authors, among them being Shurley of Detroit, and at considerable length by Arnold of San Francisco, in Burnett's first System. Newcomb, in the textbook, "Burnett, Ingals and Newcomb," speaks of abscesses probably occurring but very rarely. Casselberry, in Buck's Handbook, appears to have seen just such cases as those described in this last group, mentioning very similar conditions. When the abscess occurs on the lingual side of the epiglottis, he speaks of the inflamed condition as causing much dyspnea, but no great amount of dysphagia. Morell Mackenzie, who saw about everything that was ever observed, mentions abscess of the epiglottis as sometimes occurring.

Some of these authors speak of the matter under the general head of perichondritis of the cartilages of the larynx;

others under the head of affections of the epiglottis. Lennox-Browne said he had never seen a case of pure and simple perichondritis of the epiglottis, and mentions the fact that the fibroelastic cartilage with no very well developed perichondrium is not such as would be conducive to the production of perichondritis.

Unless we arbitrarily presuppose that a quiescent cyst was present in these two cases of pure abscess formation, and that the condition resulted in edema and pus formation, we are obliged to come back to one or two suppositions as regards their formation; either a perichondritis or an epiglottiditis. Neither of these two patients had ever had an attack of sore throat which could possibly be construed as being the same as the one described, and the existence of old branchial cysts cannot be considered.

Incidentally, a small retention cyst from the stopping of some mucous gland could be the origin of the trouble as we so often see it in an anatomically similarly constructed part of the body, viz., the eyelid, when a stye forms. In stye-formation we often have a simple Meibomian cyst existing for a long time previous to infection, and then we have what is usually called the stye, with the formation of pus following the infection. The analogy is very good here, and I have never seen a case of chondritis of the lid except from infection after an injury or infection of these cysts. It would only remain, then, to know how often these simple retention cysts of small diameter occur to make the picture complete. These, however, have never been reported. I have looked rather diligently for them since the idea occurred to me, and I have not found them as yet, but these edema cases with abscess are also very rare, these three being the only ones I have ever seen in twenty-three years' experience. So that we may very reasonably assume that one would not often find the stopped-up mucous glands which we are supposing to exist as a preliminary to the formation of abscess. Furthermore, racemose mucous glands are not numerous on the lingual surface of the epiglottis. Not giving credence to this explanation, we are driven back to the condition of idiopathic inflammation of the epiglottis, a very difficult thing to understand in these cases where no previous inflammation has existed, and yet is the one adopted by numerous writers. Ballenger in his recent book gives a separate heading, "Acute infectious epiglottitis," giving Theisen credit for the term. The latter's admirable paper had escaped us. Both these observers would evidently class such cases as the three under consideration in this group.

These three cases also differ very markedly in my observation from certain others where the edema for example, follows a simple tonsillitis. Difficulty in swallowing is the first symptom. Take for example a certain student in whom, following the mumps with a sore throat, there developed a very marked swelling and edema of the left arytenoid and aryepiglottic fold. The edema was of the baggy type, and fell over into the larynx on inspiration so that it entirely covered over the false and true vocal cords. Multiple puncture and the steam atomizer again proved of the utmost relief and speedily reduced the swelling on this side, but the next day it appeared on the other, and remained there with the greatest persistence and in very threatening amount for two whole days, when it yielded to treatment and promptly recovered.

I have had a similar experience in perhaps ten other cases where the edema followed previous inflammation in other parts of the throat and was not associated with any real perichondritis of the adjoining cartilages or inflammation of the cricoarytenoid joint. In every case it did not affect the opposite side, as in the instance related, but usually did, although only once were both sides simultaneously attacked. In these cases we have apparently a simple inflammatory affection of the mucous membrane with edema, exactly as we see it sometimes in the uvula and soft palate, with no false membrane and no other manifestations.

At present writing there seems to be good explanation for this phenomena, or for the spreading and more or less self-limitation of the inflammation. I have thought that in most of these fugacious cases there was some connection with the grippe, but was only twice able to positively connect the two. Three of the ten were certainly at a time of the year when grippe was neither prevalent nor at all probable.

No abscess formation in the arytenoid region has been noticed in my cases, except in that form of genuine perichondritis which follows severe scarlet or typhoid fever. In one instance of typhoid perichondritis there resulted the exfoliation of a small piece of an arytenoid cartilage, and in another the whole cricoid was involved and became necrotic. The one case caused complete immobility of one arytenoid, with fixation of it near the median line. The other case produced the same fixation in the median line of both chords as might have resulted from a pure and simple paralysis of the crico-arytenoideus posticus muscles. Both cords being in the median position, it was necessary to do an immediate tracheotomy. These cases belong to a very different group from the fugacious type, which latter, while they may be exceedingly threatening at the time, seldom leave any complications. One has to act quickly and rapidly in the premises, but the ultimate outcome is good. Also we have to distinguish again edema of the glottis as it occurs as a result of some systemic condition such as disease of the kidney or pupura hemorrhagica.

I have seen one such case of what one might call general edema of the glottis such as is shown in Gruenwald's Atlas as published by Saunders. This edema involved, as is shown in the picture, not only epiglottis and glottis and the interior of the larynx, but also the upper part of the trachea. In the case shown, as in my own, no operation was of any avail.

To recapitulate, then, we certainly have several distinct types of inflammation; the branchial cysts, with recurrent enlargement, edema of the epiglottis with abscess formation due perhaps to small inclusion glandular cysts or to be classed as acute infectious epiglottiditis, and then the fugitive edema of the simple or local type following inflammation in the throat, which latter is not to be confused with the two types which result from pure perichondritis, as after typhoid fever, and from the firm swelling with slight edema with which we are all so familiar as it occurs in tuberculosis and syphilis, these are all to be distinguished from the general edema resulting from systemic causes and burns or other traumata.

XXXIX.

SOME INTERESTING CASES OF DISEASES OF THE ALVEOLAR PROCESS.

By W. H. Haskin, M. D.,

NEW YORK.

Scarcely a day passes in the practice of our special work that each of us does not see new patients, each presenting some point of interest, but I think that the following are a bit out of the nose and throat area, as ordinarily practiced by us, and may prove interesting.

They were all referred to me for treatment by their dentists, who had been students of mine at the College of Oral and Dental Surgery. The question arises whether we should not enlarge our sphere of usefulness and give these sufferers the benefit of trained surgical skill rather than allow them to remain in the hands of their family dentist who, generally speaking, is not properly qualified to take care of them.

The results to the patients in the following cases were all most beneficial, but I feel that the chief good has been derived from the practical demonstration to their dentists of the foolhardiness of attempting to treat these cases through the root canals in some, of not resorting to surgical methods in all, and of the results of not being honest with them when extracting. I will give brief histories of twelve cases that have been treated by me, some of which resemble those reported by Dr. Robt. Levy, before the 1907 meeting of the American R. L. & O. Society in his paper on "Tuberculosis of the Mouth."

Case 1: Miss B. A., age 25, a teacher, was brought to me by Dr. Mayer, on May 15th, 1907. Her general health was always good. Four years before the lower left first molar had been filled with amalgam, the root canals being devitalized and filled. Since then she had never suffered any pain, but four months ago she noticed a gum boil pointing just below the gingival margin on the labial surface of the alveolar process, which broke of itself and did not cause any pain. This recurred several times until one month ago when she called upon Dr. Mayer. He removed all the root canal fillings and found that one of them communicated with the fistulous tract and then brought her to me. With a probe I found a large cavity in the process and evidence of carious bone. Under subperiosteal injection of a one per cent solution of cocain and 1:2000 solution of adrenalin chlorid I uncovered the bone at the fistula and removed all carious bone, completely exposing the two labial roots which were found surrounded by granulations. The cavity was then packed tightly with iodoform gauze, and Dr. Mayer was instructed to carefully fill the canals to their apices which were visible. He then took charge of the case and has reported an excellent result, the wound having filled in well, leaving only a slight depression.

CASE II: S. J. B., age 29, a lawyer, was brought by Dr. B. B. Machat on April 10th, 1907. The family history was tubercular, and the patient looked as though he also was a victim of that dread disease, being very thin and pale, appearing decidedly septic. For two years he had suffered with recurring attacks of pain, swelling and offensive discharge from the left upper alveolus over the canine socket, this tooth having been removed after an acute abscess about three years before. Dr. M. noticed a fistulous tract and treated it with syringing, but as it did not improve he brought the patient to me. A probe revealed a deep fistulous tract, full of granulations and areas of carious bone extending in several directions. On April 11th I operated upon the patient at the Manhattan Eye and Ear Hospital, under ether anesthesia. Here I found very extensive destruction, the caries reaching the floor of the nose and antrum and involving the whole thickness of the alveolus for at least three-quarters of an inch, necessitating the removal of the bicuspid. As there was no trouble in the antrum I carefully avoided opening into it. The wound was packed with iodoform gauze, as usual, it being allowed to remain for two days. After the second dressing I sent him to Dr. Machat for further treatment. December 14th, 1907, the doctor reported that the wound had healed rapidly, that the patient's general health had improved greatly and that a special bridge covered the depression in the alveolus.

CASE III: Mrs. M. F., age 59, was referred to me by Dr. A. Tepper, for severe pain in the upper jaw of many years' duration. Twelve years before she had had four teeth removed by a Dr. Koontz, the other twenty-eight having been removed twenty years ago, when she resided in Germany, although her husband declared that they were sound. Until one year ago she had been comparatively free from pain, but since then it had been almost intolerable and had caused her to lose much flesh, and her husband feared insanity from the insomnia. Examination of the mouth revealed a minute fistulous opening over the left lateral incisor socket which led into an extensive pocket with evidence of caries, and the whole alveolar process was much thickened in that region. Under cocain and adrenalin anesthesia I uncovered the fistulous area and removed considerable carious bone. Here, however, I found the edge of a tooth and immediately took her to Dr. Hasbrouck, who succeeded in extracting a perfect, unerupted incisor tooth, which must have extended into the nasal process, it being three-quarters of an inch long. October 20th, 1905, the patient reported by letter that all her distress had disappeared and that she was able to wear her plate with perfect comfort. This case, presenting an unerupted tooth at the fifty-ninth year, was, to me, a most interesting case and would easily have been diagnosed had the x-ray been used.

CASE IV: Mrs. M. H., age 35, was brought to me on November 1st, 1904, by Dr. S. Goldstein. The patient was very anemic, thin and septic in appearance and gave a family history of tuberculosis, although she had no symptoms of the disease in her own lungs. Two years before the upper right first molar was destroyed by caries leaving the roots. Since then she had always had more or less trouble, but never much pain until two months ago when a doctor lanced a painless gum boil. Since then the face had been swollen and there had been a constant discharge of foul smelling pus in the mouth. On examination I found the whole side of the face swollen and edematous, the right superior alveolus badly diseased, full of roots and discharging pus from just above the gingival border of the first molar root where it had been lanced. A probe revealed caries in the fistula and around the roots of several of the teeth. Under hypodermic injection of cocain and adrenalin I removed all the roots and found a large opening into the antrum at the first molar socket, surrounded by carious bone and granulation tissue. There were three distinct areas of caries at other points, all of which were thoroughly curetted. After removing the caries around the antral fistula

I could introduce my finger into the cavity, and finding it free of granulations I did nothing with it, but packed the three carious areas and the antrum with iodoform gauze. I treated the patient for two weeks and then, all swelling having disappeared and there being no discharge from the antrum, I returned the patient to Dr. Goldstein, with an antral drainage tube held in place by the packing. Dr. G. reported at intervals for three months, at the end of which time the patient was entirely well and was wearing a full upper indenture with much comfort.

Case v: A. M., male, age 46, was brought to me by Dr. W. H. de Groot on April 11th, 1906. The patient had been suffering intense pain for over a month and had had his right upper bicuspid and first molar extracted for relief, but without benefit. He then changed his dentist, and Dr. de Groot immediately brought him to me for consultation. The patient gave a history of a long standing point of discharge over the right upper bicuspid, but had had no pain until a month ago. At that time the cheek became swollen and he had intense pain which was supposed to be due to ulceration of the root of the bicuspid, and it was extracted but gave no relief. The first molar was then extracted, but again gave no relief. On examination I found the sockets of the two teeth filled with granulations, extensive caries and a large opening into the antrum through the molar socket. After removing all the diseased tissue, as I supposed, the antrum was packed with iodoform gauze. Much to my surprise, the pain was not relieved, although the wound was clean and began to heal nicely. At the end of a week I sent the patient to Dr. Sinclair Tousey, who made an x-ray negative of the alveolus for me, which disclosed, very beautifully, a pus sac involving the inner root of the second molar on the posterior border of the wound. This was easily reached, and on being opened and curetted gave instand relief. I had Dr. de Groot extract the pulp from that root and fill it. On June 4th, 1906, he reported the patient well, except for a very small opening into the antrum, which was closed by a close-fitting bridge. As there was no discharge or inconvenience caused by its presence I recommended leaving it alone.

Case vi. F. A. S., male, age 51, a resident of Syracuse, was referred to me by Dr. Demarest. He gave a history of toothache, followed by a gum boil, three months before and persistent pain ever since with a very offensive purulent discharge from the left nares. In July he had had his antrum punctured through the alveolus and had been compelled to wash it daily to overcome the offensive discharge. On examination I found the alveolar opening surrounded by granulations and carious bone, and advised operation, but as he wished to return to his home in Syracuse I referred him to Dr. Halstead and have not heard from him again. This was undoubtedly an antrum suppuration prolonged by the carious bone at the alveolar opening, and would have been easily cured by its thorough removal of the caries.

CASE VII. J. B., male, age 46, a blacksmith and a resident of Sidney, N. Y., was brought to me by his dentist, Dr. A. P. Clarke, on October 23rd, 1904. Nine months before he had had the left lower second molar extracted, although it was sound, to try and obtain relief from pain apparently located at that point (the first molar had been extracted some months before that). Following this the cheek became swollen, and he had had a partial facial paralysis for a few months. Some time later a swelling appeared under the lower jaw, which broke under the tongue and gave no further trouble. Ever since then there had been a hard, painful mass on the jaw, which at times discharged pus and was gradually increasing in size. The pain had grown more and more intense, causing loss of sleep and much interference with mastication. On examination I found a very hard tumor, about the size of a large hazel nut, with a minute fistulous tract leading down to exposed carious bone, and I advised operation. He was anxious to return home and insisted upon immediate action, so under hypodermic of cocain and adrenalin I uncovered the mass, which appeared to be a dense osteoma, and removed it with chisel and bur. As I was smoothing the cavity with the bur, thinking that all trouble had been removed, the instrument struck a harder mass, which proved to be a broken root of the first molar. This had probably been the cause of all the pain, and would have been disclosed in an x-ray plate, and thus have saved the sound second molar, removed as a possible cause of the pain. Dr. Clarke took full charge of the patient at his home, and three months later reported that he had made a rapid recovery and had been entirely free from pain from the time of the operation.

CASE VIII. Miss J. C. B., stenographer, of N. Y. C., came to me on June 9th, 1905. She was about to have a new full upper indenture made, but her dentist was afraid that it would not be a success, because of an irregular thickening of the alveolus on the left side at the site of the former canine and bicuspid teeth, it being also somewhat tender on pressure. At first sight I did not notice anything save a pin head depression on the labial surface of the thickened alveolus, but on using a Buck's ear probe with some pressure, I found that there was a fistulous tract filled with granulations and that there was carious bone present, although she insisted that there had been no discharge at any time. Under cocain and adrenalin hypodermic anesthesia I uncovered the area and was rather surprised in finding extensive caries involving the whole thickness of the alveolus and extending to the floor of the antrum. The cavity healed rapidly with iodoform packing changed daily, and at the end of a month she was able to be fitted with her new indenture, which she has worn with comfort ever since.

CASE IX. Mrs. B., age 40, of N. Y. C., came to me in January, 1908. Her general health was good, but for over a year she had had a small fistula over the socket of the right upper canine, which had been extracted, from which she could always suck out an offensive pus, but it was never painful. The tooth had been extracted and the fistula treated by her dentist, but it had never closed. A probe revealed carious bone and a tract three-quarters of an inch in depth. Under local anesthesia by hypodermic I uncovered the tract and with bur and chisel removed all the diseased tissue, packing it with iodoform gauze, as usual. At the end of five weeks there still remained a small area of exposed bone, which I pared down with the bur, and the wound then closed completely and has given no trouble since.

CASE X. C. P., male, age 47, a printer, was referred to me by his dentist, Dr. A. Tepper, on June 4th, 1907. He complained of intense stabbing pain in the left upper alveolus, and had had all his upper teeth extracted, but without any relief, the pain steadily increasing. As I was leaving the city two days later, I told him that I would not attempt any treatment

During the summer he visited three physicians, but after trying many medicines he refused to take opium, fearing lest he contract the habit. On my return in September he promptly made his appearance, complaining bitterly of the pain, which prevented sleep and mastication and was located at one spot where the first molar had been. I examined the alveolus very carefully, but found absolutely nothing to account for the pain. An x-ray photograph was taken, but disclosed no root or evidence of caries. At his urgent request I uncovered the alveolus under hypodermic injection of cocain and adrenalin, but without much hope of locating the cause, believing it to be a case of trifacial neuralgia. On elevating the periosteum I found a curious condition, the socket of the first molar having closed below still remained of almost normal size, was full of granulation tissue and opened into the antrum. I was greatly surprised at this, as I had puctured the antrum through the nose and irrigated it with a solution of boric acid, but got no pus, and the patient declared that he had never had any nasal discharge. The whole thickness of the process was softened and bled very freely until I reached healthy bone, in doing which I had to enlarge the antral opening until I could easily introduce my finger and examine its interior which was found healthy throughout. At one point he exclaimed that I had touched the exact spot, but I saw nothing to account for it. The antrum and cavity were packed with gauze, it being changed daily. For ten days there was almost complete relief, but the pain then returned and was located by him at one spot on the anterior border of the cavity. After cauterizing this, without relief, I again used the bur and opened into another pocket (the bicuspid) filled with the same dark red granulations, which bled profusely, so that I sent them to the laboratory for examination. The report received, however, was that they were granulation tissue with many capillaries but showed no evidence of malignancy. The last operation brought permanent relief, at least I hope that it has, for there has been no pain from November 5th, 1907, to April 10th, 1908, the date of his last visit which he made to pay his bill and give thanks for his cure. He was wearing his old indenture with comfort, and objected to my suggestion to close the antrum opening, which was the size of a small lead pencil, saying that there was no discharge from it, and as it gave him no trouble he thought he would let well enough alone. I was afraid that this case of tic doloureux was caused by malignant growth, because of the most unusual severity of the pain and the hemorrhagic character of the granulations, and am happy indeed at the result thus far obtained.

CASE XI. Under the title of "An Inflamed Dentigerous Cyst Caused by an Odontoma," I reported a case before the American L. R. & O. Society at its annual meeting in 1905. This case illustrated the folly of the indiscriminate extraction of teeth for the relief of pain before using all available means to determine just where the trouble lies. In this case the original neuralgia was probably caused by the odontoma, and had the dentist done his work completely and not allowed the broken root to remain, he would, in all probability, have cured the neuralgia and removed the undoubted cause of the cyst in later years, at the very first extraction, the original pain having been located by the patient in his bicuspid, the root of which was found by me. I asked for an x-ray in this case, but, being a poor man with a large family, he could not afford the expense and it was really only by chance that its presence was discovered.

CASE XII. This case was also reported by me in full before the 1907 meeting of the American L. R. & O. Society, under the title of "A Case of Fibro-mxyoma," etc. Had this case been seen originally by a qualified surgeon I do not doubt but that thorough surgical measures would have removed the disease entirely and secured a complete cure, thus obviating the further suffering and the very extensive later operation.

The foregoing cases only serve to illustrate the condition of many thousands of patients seen by us but not recognized, our attention not being called to what they believe belongs strictly in the province of dentistry. Unfortunately, there are very few dentists, in the great numbers of those licensed to practice, who are qualified, either by experience or in their college education, to recognize the extent of these cases or the seriousness of allowing them to run on indefinitely. There are, alas, too many who will persist in treating these conditions through the minute root canals. As professor of oral surgery in the College of Oral and Dental Surgery of New York, I saw a great many cases of these fistulous tracts, and in all cases of long standing advised the free opening of the alveolus and the removal of the carious bone wherever found either with or without the extraction of the teeth, that depending upon the condition of the bone immediately surrounding the tooth. Often it will be found that only one root will be entirely uncovered, and by amputating this and carefully closing the canal entrance the remainder of the tooth can be saved to be very useful, the carious tract being cured. It is absurd to say that it is best to treat these cases through canals when one has once seen the extensive destruction of the bone that will be found in all long standing cases, and we should urge that surgical measures be used in all cases where there has been a so-called gum boil, which fails to heal and leaves a fistula, however small the opening may be, for they are in reality cases of suppurative alveolitis. which have opened through bone tissue and left disease of that tissue, which naturally cannot be reached through the tooth itself.

There are also many cases where teeth have been extracted after long treatment without benefit, and no examination of the socket is made to determine whether there is caries or detached spicula of bone, and. although in some cases Nature succeeds in asserting herself and throws off the dead bone, many of these still retain their discharging fistulas, much to the annoyance of the patient. Another point which I wish to emphasize is the necessity of having all of our friends and patients who have to suffer extraction, either bring the tooth to us or have the dentist report direct, where we know the operator. There are too many cases where the tooth is broken in the attempt and the root is allowed to remain to be the focus of further trouble. I have no doubt that the cases 7 and 11 would have been relieved by the first extraction had it been complete and the dentist honest in his dealing with his patient, but one continued to suffer for over a year, lost a second sound tooth and had to have an osteoma removed in consequence; and the other lost all of his upper teeth and had to undergo an extensive operation for the removal of the large bone cyst in consequence of retention of the broken root with its large odontoma. which was in all probability the cause of the original pain and of the development of the cyst. The more one observes the condition of the teeth in his laryngeal and pharyngeal cases the more will he be impressed with the great benefits to be obtained by attention to oral hygiene so sadly neglected in many of them. In recent years the dental profession is awakening to the fact that these conditions have a much more serious import than had been given them through many years and they are slowly developing a specialty among themselves, whose members devote themselves to the treatment of surgical cases only, and I am happy to say that much excellent work is being done by some of the men that I know, and that two of the men who brought their patients to me have since done some very praiseworthy work and are most grateful for the opportunity afforded them in seeing what could be done for the relief of what had been to them difficult cases to treat and almost impossible cases to cure. With our experience in surgery of the nasal cavities and especially the mastoid process, the operation for the removal of caries in the alveolus, is very simple and in only one case was the patient anesthetized with a general anesthetic, i. e., Case 2, he having such a large cavity at the examination that I felt it wiser to be prepared for any condition that might have arisen. As it turned out, the operation could have been performed with the local anesthesia very comfortably. Dr. Sinclair Tousey has made some very satisfactory negatives for me by his method of placing a small film on the inner surface of the alveolus, thus taking only that part of the process in which the trouble is actually located, so that there is no confusion in locating roots or caries if they are present.

XL.

SUBMUCOUS RESECTION IN OZENA.*

By Prof. Mermod,

LAUSANNE.

It is well known that ozena may be unilateral, a fact which is naturally regarded as proof of its non-contagiousness. But no one, I believe, has set forth that where the ozena exists on one side only, it is constantly the larger side which is attacked, and that in very marked deviations of the septum the process is limited to the concave side.

Personally, at least, I have never found a unilateral ozena where both fossae were of the same size, and, on the contrary, in every lateralized ozena I have regularly found a septal gibbosity and the atrophic rhinitis on the larger side. Where the deviation is not pronounced the ozena is bilateral, but less marked on the narrow side.

This finding is so frequent, so striking, and its etiologic importance so great, it is surprising that it has not been made before, after all that has been written upon the origin of atrophic rhinitis, fetid or non-fetid. If the fact which I advance has already been observed by others, I do not think that it has been accorded the attention which it deserves.

What do we see in typical cases of this sort? I mean those in which the curvature of the septum is sufficient to efface one naris. On the enlarged side is the well-known picture of classic ozena, dryness, crusts, fetor, atrophy of the inferior turbinate, atrophy or hypertrophy of the middle turbinate. On the narrow side is ordinary catarrhal rhinitis, due to mechanical obstruction, without crusts or atrophy, but with a mucous catarrhal secretion coming forward or into the cavum. Objection cannot here be made that it is not surprising for the narrowed side to be without crusts, since the necessary space is lacking, for there is more than an absence of crusts, the dryness and atrophy also being lacking. The subjective signs correspond to the objective. The patient breathes as badly on one side as the other, since he cannot distinguish between obstruction due to bending of the septum and that caused by the accumulation of crusts, but he does state that he blows green crusts from only one side and ordinary mucus from the other.

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*Communication to the International Congress of Laryngology at Vienna, April, 1908.

It goes without saying that if the malformation of the septum involves only its anterior portion in the form of a spur or luxation of the anterior border of the quadrangular cartilage, leaving a nasal fossa as large behind as its fellow, the ozenous process involves both sides.

The etiology of a disease as complex as ozena has necessarily given rise to innumerable theories. The only one which interests us here is that where, according to Zaufal, we see in congenital enlargement of the nasal fossae, the essential cause of ozena. Inspired by this idea several investigators have sought with more or less success to diminish the size of the fossae, either by tamponing the whole naris (Gottstein) or by anterior occlusion with tampons or obturators, or by plastic operations (Barth and Saenger), or by injections of paraffin.

Now, if any one of these means would cure ozena with certainty, the theory of congenitally large nasal fossae would acquire sufficient importance to be something more than a theory. Unfortunately, this is not the case. However, the interesting fact of lateralization of ozena in pronounced deviation of the septum appears to me to be the most convincing proof that has ever been brought to the support of Zaufal's theory. But, I hasten to add, this proof does not apply in special instances, since in most cases of septal curvature the mucosa of the enlarged side is not only not atrophied, but shows a compensatory hypertrophy, so that sometimes the septum appears to have been pushed aside by the thickened inferior turbinate. From which it is to be concluded that increase in size of one or both nasal fossae, as is well known, can only be a predisposing cause of ozena allowing other factors—microbes, suppuration of accessory cavities, trophoneuroses—to act more effectively.

But well observed facts ought to take precedence over theories, and if, gentlemen, I have succeeded by straightening the median septum in suppressing the cardinal symptoms of ozena, you will agree that this thesis is of more value than you suspected.

If with a good submucous resection I reestablish equality of the nares, I kill two birds with one stone, if I may thus express myself, by suppressing on the narrowed side the simple catarrh due to obstruction, and on the enlarged side the ozenous catarrh. For this it is necessary to make a perfect resection, which is not difficult, the mucosa of the internal wall being much less atrophied than that on the turbinates. The persistence of a septal perforation, however small, while of little importance in the ordinary cases where we do this operation, would here have deplorable consequences, as is readily understood.

I submit to you some cases which are so similar that I can condense them into a few lines.

1. Michel S., Russian, 10 years.—A puny child, adenoid facies. Has always blown a good deal from the left nose, especially since his fifth year (scarlatina). The secretion is purulent, greenish, mixed with crusts. Frequent catarrhal exacerbations during which especially the mother notices the fetor, which leads her to bring the child to me. External nose shows nothing peculiar. Left naris very large, plainly ozenous. The upper part of the naris and middle turbinate, which is large and baggy, are covered with greenish dry crusts, hard to get off. The floor of the nose is bathed in thick mucopus; inferior turbinate greatly atrophied. No sign of sinusitis. Septum much deviated throughout. Right naris very narrow; middle turbinate invisible; inferior turbinate of normal size, touching the septum; mucous secretion, no crusts. Submucous resection. When seen three months later the child seemed absolutely cured, objectively and subjectively. The left naris had a red moist mucosa, without crusts or odor, although the mother had ceased the daily lavages after the operation. Inferior turbinate had a thicker, red, moist mucosa. General condition much improved.

2. Alfred M., Swiss, 17.—Marked deviation of the septum closing the left naris. The right is very large, because of the septal concavity and atrophy of the inferior turbinate. Middle turbinate big and soft, fetor. Not much discharge, but the secretion is muco-purulent, mixed with greenish crusts, covering the external wall and roof; twice a week they are expelled *en masse*. Pharynx similarly covered with crusts—dry atrophic phanryngitis. Immediately after submucous re-

section the result was excellent. Diminution and then disappearance of the crusts, so that the young man no longer used the lavages, which he could not do without previously. Middle turbinate remains large, but less soft, and no longer bleeding at the slightest touch. The inferior turbinate, lubricated and red, seems less atrophic. The cavum still has a little pus, but not drying into crusts. Pharyngeal mucosa remains dry.

3. Alfred M., Swiss, 18.—Atrophic rhinitis from earliest years. Three years ago I taught him to irrigate his nose, and for a long time used friction massage with sulphoricinated phenol. Seen again last January 2. Typical picture of fetid atrophic rhinitis. The family is annoyed by the odor. The process localized solely in the very large left naris, made so by the septal deviation. All walls covered with thick greenish crusts separated from the atrophied mucosa by a layer of muco-pus. Inferior turbinate reduced to a thin ledge. especially the anterior portion. On the right side the septum touches the inferior turbinate which is not at all atrophied—no crusts, only catarrhal mucus. Respiration habitually buccal; dry pharyngitis. The voice is frequently interrupted, the vocal cords being slightly crusted without there being exactly a laryngo-tracheal ozena. Resection of the septum done the following day. The results as seen three months later could be called brilliant. The family is particularly delighted with the absence of fetor, although lavage has been stopped. The mucosa is still atrophied, but moist, and without any crusts. Larynx all right and voice clear.

4. Alice F., 22, is also one of my old patients, whom I have given various treatments without success, except that by great care she had lessened the intensity of her trouble. She suffered especially with fetor, and, as she was about to become engaged, her repugnant infirmity made her quite melancholy. It was another typical case of left unilateral ozena with pronounced deviation of the septum to the right. Believing that the cause was an ethmoidal caries, I did several endonasal curettages without any appreciable improvement. I showed the case to the well-known author of "The Coccobacillus foetidus ozenae," my friend, Dr. Perez, of Buenos Ayres, then sojourning with me, and he did not hesitate in the diagnosis of typical fetid ozena. I shall not recount the

subjective signs, which were exactly the same as those in Case 3.

After the submucous resection the reaction was intense, and during the first month the crusts were so abundant and thick that I thought I had failed absolutely. Then came progressive improvement. I saw the patient a fortnight ago, three months after the operation, and had the pleasure of seeing an extraordinary amelioration. Not only have the crusts ceased to form, but the mucosal atrophy, especially that of the inferior turbinate, has so diminished that it is hard to believe in the existence of a former ozena. The patient has given up the lavages, which were a daily necessity and were, she says, "the cross of her life."

These, gentlemen, are the cases which I have had during three months, in which, by the simple suppression of a mechanical fault, the effects were invariably good, notwithstanding a prognosis so unsatisfactory as that of ozena. And I do not think that I am too enthusiastic over the results, which have been watched by my medical associates, assistants, pupils and visiting colleagues. Of course, we cannot speak of a definite cure as yet, and the time is too short since operation to know the final result, but the example of my fourth case permits the hope that with a disappearance of secretion and the atrophying pressure of the crusts upon the mucosa having ceased, the latter will again gradually take on its normal character.

Results less affirmative than mine would not detract from the latter, the negative cases not having the value of positive ones. They simply prove once more that ozena, far from being a morbid entity, is a complex not yet well understood.

It would be fortunate for my assertions to be further confirmed, since they touch upon an important etiologic and therapeutic question.

XLI.

THE TREATMENT OF TUBERCULOSIS OF THE UPPER AIR PASSAGES.

By Dr. Theodor Heryng,

WARSAW.

Translated by Clarence Loeb, M. D.,

ST. LOUIS.

In my paper I will omit reference to tuberculosis of the mouth, pharynx, nose and nasopharynx, and will discuss only the treatment of tuberculosis of the larynx.

The following prefatory remarks will make the matter clearer:

Larynx tuberculosis, or the phthisis of the larynx caused thereby, can heal spontaneously, without special, local or general treatment.

Most frequently, superficial ulcers of the vocal cords and milder cases of affection of the posterior wall of the larynx and processus vocales heal by cicatrization. Severe, destructive processes, which are accompanied by granulation formation and lesion of the cartilage, followed by dysphagia and consequently rapidly leading to malnutrition and cachexia, very rarely heal, especially in young patients and those affected with diffuse pulmonary affections, accompanied by fever and lowered body resistance.

The best chances for healing is found in those cases where the disease appears as a localized infiltration or tumor-like prominence on the posterior laryngeal wall, which has little or no tendency towards degeneration, causes slight symptoms and is accompanied by no apparent loss of bodily vigor. Here belong, also, the so-called tubercular tumors of the larynx, especially of the false cords.

The worst prognosis is to be given in the diffuse infiltrations of the outer laryngeal borders, viz., the epiglottis, when they extend over onto the false cords and attack the ventricle; furthermore, infiltration of the arytenoid cartilages, which leads to rapid destruction and necrosis, or when accompanied by granulation formation cause stenosis; also, the diffuse, degenerative infiltrations, involving the larynx in toto; finally, miliary tuberculosis.

The possibility of a cure is, in the first place, dependent on the constitution and resistance of the patient, the anatomic character of the pulmonary affection and its extent, the age of the patient, his occupation, his financial status, and the period at which the proper treatment is begun.

Although the tubercular process may heal, or cicatrize, tubercular deposits may remain in the depths of the tissue, which, although subject to fibrous degeneration, possess quiescent germs, which can cause recurrences. This occurs so much more easily if renewed unfavorable conditions, such as straining, cause inflammations, which lead to dysphagia and malnutrition. They almost always lead to exacerbation of the pulmonary lesion. Secondary infections play in laryngeal phthisis the same weighty role that they do in pulmonary tuberculosis.

Of all the various drugs used in laryngeal phthisis, such as creosote-glycerin, phenol, balsam of Peru, sublimate, iodoform, iodol, menthol, etc., the only one that has gained a distinct position in the therapy is lactic acid, introduced by Krause.

This remedy cannot, however, be used in all cases, as it often causes severe inflammatory symptoms. It is indicated in hypertrophic ulcers, in soft granulations, and for the purpose of cleansing the floor of ulcers. It has very little effect on hard infiltrations.

Menthol and iodol are far behind lactic acid. Iodoform is indicated in certain forms of ulcers for cleansing and for excitation of granulation formation.

Phenolum sulforicinicum frequently exercises a favorable influence on tubercular infiltrations, but requires certain precautions in its use.

Cocain and eucain may be used as sprays in 5 to 10 per cent solutions by means of a curved tube, but only during phonation, in order to prevent poisoning.

Antipyrin lengthens the anesthetic action of cocain and eucain (Weil). Orthoform, advocated by Einhorn and Heinz, act well in the dysphagia of larynx phthisis. It acts, however, only on ulcerated surfaces denuded of epithelium, and is absolutely contraindicated in infiltrations. Its action in ulcers lasts from 6 to 10 hours. It is advantageous to prescribe it with an equal amount of talc and an addition of one per cent cocain (or eucain) to 4.0 of orthoform. The insufflations should be repeated twice daily. The patient must be forbidden to gargle or drink afterwards.

For about six years I have been induced by the powerful, deep action of concentrated potassium permanganate solutions in lupus to use it for the destruction of unclean tubercular ulcers, which constantly renew their dirty gray exudate, and the results have been excellent. Such conditions were most frequently found on the infiltrated and ulcerated false cords, never on the true cords or posterior laryngeal wall or epiglottis. This exudate consisted of necrotic tissue filled with innumerable pus cocci, epithelium and detritus. Tubercle bacilli were never found. None of the drugs in use could destroy this exudate. Even after the use of the galvanocaustic and the formation of a scab, the exudate reappeared on the granulating wound. Only after repeated painting with a lukewarm, saturated solution of potassium permanganate did I obtain healthy granulation formation and a slow cicatrization. I judged that the process affected the mucous membrane of the ventricle also, and from there passed over to the false cords. Further observations showed the favorable influence of this remedy on chronic ulcerations and even superficial infiltrations, for which it can be most heartily recommended. Its use is painless, the reaction is nil. The black scab is cast off in two to three days, according to the intensity of the cauterization. Never allow crystals to remain on the swab. The action is very deep, the tissue melts away by a quick necrosis and a deep defect remains. I have observed this deep action in pharyngeal tuberculosis and warn against the use of crystals, on account of the severe pain which they cause.

The solution is made best in a reagent glass, heated to boiling, the crystals pulverized by a glass rod, and the fluid carefully decanted from the precipitate and cooled. The swab must have no excess of the solution. Paint it until the ulcer has a very dark color. The dysphagia frequently becomes less after the cauterization. The infiltration shrinks and becomes covered with new granulations after the scab is cast off.

I have used the same drug in nasal tuberculosis, lupus and syphilitic nasal ulcers, frequently with quick result. It is, however, no panacea, for I had, also, cases without results and had to use surgical treatment.

The treatment of laryngeal phthisis is divisible into two groups, according to the nature of the disease, i. e., the treatment of the laryngeal lesions, and that of the pulmonary, which almost always complicates it.

In a disease which has lasted many years, it is necessary for the patient to learn that improvement and eventual cure is obtained by prevention of the unfavorable conditions which have caused it.

The localities along the Mediterranean, of the Riviera or Levant, although they have a lime subsoil, have in many cases been found to be satisfactory (Mentone, San-Remo, Cannes, Nervi). Favorable results have also followed residence in Meran, Arco, Palanza, and, in summer, Gleichenberg. According to my mind. the choice of the place is far less important than the choice of the physician. Weak patients with high fever, severe dysphagia and extensive pulmonary involvment, and who are poor, should not be sent to these far away places.

The first condition for the improvement of these patients consists in access to an abundance of pure, fresh air.

Feverish patients, so long as their strength allows, are kept in bed, in order to conserve their strength and prevent discomfort and weakness.

The partial, temporary and frequently insufficient results of the treatment with lactic acid, as well as those of the incision in the infiltrated epiglottis or posterior laryngeal wall, as recommended by Moritz Schmidt, and which in some cases causes a diminution of the dysphagia by means of a stretching of the tissues, compelled us to seek other remedies and methods which could produce positive and lasting cures. For this reason I proposed, in 1888, a surgical treatment of larynx phthisis, and published its basis in my book "Die Heilbarkeit der Larynxphthise und ihre Chirurgische Behandlung."*

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*The Curability of Phthisis Laryngis and Its Surgical Treatment.

Under the surgical treatment I had the following to say: "Above all, the attempt must be made by means of the most energetic destruction or removal of the tubercular infiltrate and its products, to get rid of everything that is diseased or degenerated, and to change the ulcers into benign ones and aid their healing; furthermore, by causing the cicatrization of these ulcers and the cure of the accompanying inflammation, to successfully combat the dysphagia and to limit the imminent dangers. A therapeutic factor of no less importance consists in the building up of the body, in the prevention of all untoward conditions, and in the securing of good climatic and hygienic conditions."

It is, of course, self-evident that no method and no remedy can be considered as a panacea for any disease, especially such an unreliable condition as larynx tuberculosis. The cicatrix obtained and the lasting cure must be regarded not so much as the result of one certain treatment, but as the result of relatively complicated methods composed of the most varied therapeutic and hygienic rules.

Their result is no stable one, but is dependent on their energetic employment, and is obtained only in certain forms and stages of the disease, under especially favorable circumstances. Although I lay a great stress on the technic of the surgical treatment of phthisis laryngis and insist upon this always, I am far from believing that a perfect technic is all that is necessary to treat the patient successfully.

The physician must be therapist and surgeon in so far as the local treatment has a good prognosis only when accompanied by care of the general condition, the nutrition, and the pulmonary affection.

The statements mentioned here were made by me twenty years ago. I have been true to them in deed and word. They seem unknown to many of my colleagues, and therefore I repeat them here to prevent ungrounded attacks on the person and the method.

I have been accused of a too great optimism in regard to the results of surgical treatment and a too great enthusiasm in carrying out this method.

I should like to quote a sentence from Kant: "Enthusiasm is that property of the human mind without which no good is ever accomplished."

It is a peculiar fact that this accusation has come chiefly from those of my colleagues who have had either little or no experience in this direction, and who, after a few usually unsuccessful experiences, have abandoned it. Some of them probably used the method in unsuitable cases. Others were discouraged on account of the difficulty of the technic, a somewhat severe hemorrhage or the use of badly chosen instruments.

In a disease considered twenty years ago as incurable, whose energetic treatment was regarded as a medical mistake (Stoerk), an unnecessary handling of infiltrations (Schroetter), or as a torment to the patient, it was a bold proposition to suggest a surgical treatment. The first cases published by Krause and myself were characterized as diagnostically uncertain. We were asked for cures of the gravest cases, patients with cachexia and diffuse infiltrations and ulcers. Every lack of result, every relapse, every passing exacerbation of these unreliable cases was used as a weapon against the method and its discover, and in certain clinics and hospitals were demonstrated to students and physicians as warning examples of the perniciousness of this method, which was condemned.

Gradually, through the aid of many trustworthy colleagues, this judgment was destroyed and the observation of more lasting cicatrization and cure of even severe cases gave an impetus to the energetic treatment (Schech, Keimer, Gleitsman, Gougenheim, Balzer, Massei, E. Fraenkel, Tessier). S. Srebrny in 1897 published a careful collection of all cases published between 1887 and 1897. Only eight authors had opposed this method, some of whom did so without personal experience and on purely theoretic grounds.

At the meeting of the Tenth International Congress in Berlin, 1880, I demonstrated a patient who had been cured for two years of a severe laryngeal phthisis. Furthermore, I showed the larynx of a patient who had been cured for three years, but who had subsequently died of intestinal tuberculosis; also, a number of cases where through surgical intervention, i. e., curetting the ulcers, or removal of the tubercular infiltration with sharp forceps, permanent cicatrization or improvement took place in the larynx. Most of the members were convinced of the correctness of my deductions. These were very forcibly supported by Moritz Schmidt and Hermann Krause. However, we were unable to overcome the opposition despite the fact that the specimens were recognized by Prof. Virchow as completely healed. Since certain members demanded a microscopical examination for the purpose of determining whether tubercular foci were present in the deeper tissue, under the scar, the specimen was sent to prosector Eugen Fraenckel at Hamburg. The result of his examination ran: "There was not even a trace of anything that could have been called a tubercle or like one."

Four years later the question of surgical treatment of larynx phthisis was again discussed at the Eleventh International Medical Congress at Rome. Gougenheim (Paris), Lennox-Browne (London) and the author were chosen as essayists. Eighteen members of the laryngologic section took part in the discussion. The result of the very earnest debate, which took place under the presidency of Dr. F. Semon, was a very favorable one for the value of the surgical treatment. Fifteen members argued for it on the grounds of personal experiences.

Just as favorable were the reports of Lennox-Browne and Gougenheim, who had used the method in fifty-eight cases and had obtained a cure in twenty-five cases and a distinct improvement in thirty. I discussed then the subsequent course of the cases cured since 1887, and presented to the section a colleague from Dynaburg, Dr. Sczastny, who for four years had been cured of a severe diffuse laryngeal phthisis, had recovered voice and deglutition, and showed such an improvement in his general condition and the pulmonary process that he could readily fulfill his office as chief physician of a large military hospital.

A distinct change has in a few years come over the position as to the surgical treatment of phthisis laryngis. Burger says that "after the original recommendations of Moritz Schmidt and Heryng had met and withstood a violent opposition, there appeared a stage of recognition, whose celebration at the Eleventh International Congress at Rome in 1894 will always be a source of pleasant recollection. At that very time, as Krieg correctly observes, the advocacy of energetic method was at its zenith. Since then it has declined, and I was afraid that Kronenberg was too optimistic when he thought that now since the enthusiasm had passed away, a sober judgment would rule, which would be as far removed from doubting despondency as from thoughtless overvaluation of our ability and knowledge."

"Like Krieg," continues Burger, "I have gained from the literature of the last few years the impression that a large number of laryngologists have confined themselves to the use of antibacterial and disinfecting remedies, that most of them use lactic acid, and that relatively few treat suitable cases perseveringly and intelligently according to the principles of surgical therapy.

"With such facts in mind, one asks in astonishment whether the earlier enthusiasm was founded only on idle dreams and whether the well-known cures were only the result of an uncriticizing enthusiasm." Burger then addressed himself to those colleagues who, in his own words, broke ground in this field, and who had for the longest time carried out the treatment, viz., Moritz Schmidt, Krause and myself.

He received from M. Schmidt the following statement: "Almost one-fifth of the cases were cured. Part of them suffered from relapses, especially when the lungs were not cured." Schmidt, however, has observed for years patients whose larynx and lung affections have been cured, and who were not reinfected.

Five months ago I inquired from Prof. Spiess, of Frankfurt, the worthy co-worker of Moritz Schmidt, as to the number of curettements made by them and the details of the results of their treatment.

Prof. Spiess estimated the yearly number of currettements at about 400, i. e., at about 6000 for the last fifteen years. He authorizes me to state here that he curettes energetically, excises and works with cutting instruments without having observed that new infections were caused thereby. On the contrary, he ascribes to this therapy a large number of cures.

Krause also, says Burger, still sticks to his earlier optimistic standpoint. He is convinced (*Monatsch. f. Ohrenheilkunde*. 1904, p. 381) that this method "would be as successful to anyone, who used it intelligently and with a proper technic," as to him.

When I learned that Krause could not take part in this congress, I went to him at San Remo, and am authorized to communicate his views.

He estimates the number of curettements performed in his policlinic and private practice at about 4000. He had never observed a secondary infection. Even in the worst cases he saw an astonishingly rapid closure of the wound.

Krause has not discarded the galvanocaustic treatment, as it is very convenient in many cases which are inaccessible except with difficulty with the surgical instruments. Furthermore, he does not doubt, though personal experience is lacking, the good results of Krieg, Mermod, etc. He cannot, however, regard this method as a panacea, which could only be claimed by one who is autocratic enough to be impatient with any method save his own. According to Krause, the galvanocaustic is only an aid to the radical surgical treatment.

In his second article, published in 1904, in the *Arch. f. Laryngologie,* Krieg says that lately he had successfully replaced the purely surgical treatment by the galvanocaustic, or had used them combined. Out of over 200 cases Krieg had obtained a permanent cure, i. e., cicatrization, in 60 poor patients, without any cauterizing agent or lactic acid. Occasionally he saw granulation formation in the vicinity of the cicatricized wound, which he regarded as an expression of the vulnerability of the tissues over the deeper, infiltrated areas.

That these results were not temporary is shown by the following letter sent to me by Krieg in May, 1908:

Dear Doctor:—

To your questions I can reply as follows:

1. I have curetted 73 times.
2. I have never had a dangerous hemorrhage.
3. I have never seen a succeeding miliary tuberculosis, nor have I observed a dangerous reaction follow the galvanocaustic.
4. I have seen 48 cases where the cure of the laryngeal tuberculosis lasted for three years. It must be stated, moreover, that an inquiry, which I have not conducted since 1904, would doubtless reveal other cases as cured.
5. An indication for total extirpation of the larynx never was seen by me.

Very sincerely,

KRIEG.

The same year there appeared the great work of Mermod

(Lausanne). By the use of sharp forceps or the galvanocaustic, he obtained lasting cures in 60 cases of diffuse phthisis of the lung and larynx of the severest type. He was able in 1903 to present 18 cured patients before the annual meeting of Swiss physicians. Of 240 cases of larynx phthisis, the cure lasted

in 60 over 1 year,
in 40 over 2 years,
in 17 over 3 years.

Even in the most severe cases, which died later on from their lung affection, Mermod obtained distinct improvement of the voice and deglutition.

That Sir Felix Semon, whom Grünwald designated as one of the opponents of surgical treatment, must be removed from their ranks is shown by the following extract from his letter of May 28, 1908:

"I think that I have clearly enough stated that we should be thankful to you for your untiring advocacy of surgical intervention in appropriate cases. I, myself, always operate in such cases, only I cannot go so far as Prof. Mermod, according to whose own words, there is no contraindication for the use of the galvanocautery in laryngeal tuberculosis. I have expressed myself in the same way in all of my writings."

We will now let the opponents of the purely surgical treatment (sharp instruments) speak, and will now commence with Grünwald. His successful results from the use of deep galvanocauterization have led him to call this method the sovereign one, and to discard the purely surgical method for fear of tubercular and purulent infection.*

Grünwald designates most of the remedies hitherto employed against laryngeal tuberculosis as unsuitable, especially the chemical cauterants, the cutting laryngeal instruments, but more especially the tearing and crushing ones, on account of injury through unnecessary and dangerous superficial traumatism and insufficient deep action. He fears to lay bare simultaneously tubercular and normal tissues—whether it be the blood or lymph system—by which a local or even a general dissemination of the infection might be caused (p. 37).

"This danger can be prevented only when this excision takes place in the healthy tissue outside of the diseased area, or when the treatment leads to occlusion of the paths of infection or to the death of the germs. For this reason alone should protection of the surface be our motto. Only the smooth excision of more or less pedunculated parts would cause no damage. Here the loop comes into consideration."

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*Die Therapie der Kehlkopftuberkulose mit besonderer Berücksichtigung auf den galvanokaustischen Tiefenstich.

According to Grünwald, the galvanocautery—the deep incision—must be considered as a panacea, on account of its slight reaction, since it gets rid of the diseased tissue and the bacteria quickly and completely, and makes possible desired result, since the infiltrate extends beyond the recognizable borders of the focus.

As to the method, Grünwald uses about a dozen lines in its explanation:

"The burning point is placed on the selected spot, and after it has been heated, is slowly pressed into the tissues, until either the distinct resistance of the normal tissues or the reddish gleam in the subglottic space calls a halt. The point should act until it is movable in the puncture-canal, about 5 to 10 seconds. Not more than two punctures should be made on account of reaction which will follow. In general, all symptoms disappear in 8 to 14 days; the scab disappears unobserved. When the burn is severe, there is a large sloughing of necrotic masses, followed by a negligible swelling."

A complete discussion of Grünwald's interesting article would transgress the limits of this paper, and will be done elsewhere. The chief point of his theory, the necessity of preserving the epithelium of the tubercular infiltration and the warning against the purely surgical method on account of infection, these have been shown by Schmidt, Spiess, Krause, Gleitsmann, Besold, Gidionsen, and by my observations of 20 years as not substantiated by clinical findings. He is right, however, when he says that infiltrations which for a long time resist degeneration, are circumscribed and exist in relatively well nourished individuals, free from fever and without dysphagia, may be treated conservatively, especially in patients who are living in sanatoria or health resort clinics. The number of curettements performed by the above named colleagues is over 12,000. According to oral and written testimony, not one of them has frequently had a severe hemorrhage or tubercular infection following a surgical intervention.

I will now take up the views expressed in a very interesting article, published this year by Besold and Gidionsen, entitled, "Pathologie und Therapie der Kehlkopftuberkulose." I can review here only the treatment, and that briefly. The interesting microscopical findings deserve great consideration, especially the photographic reproductions of microscopic specimens.

Their material consisted of 500 cases of larynx tuberculosis treated during the last 10 years at Falkenstein a. Taunus.

Of all the different methods of treatment, the surgical was found to be the best in the severer cases of larynx phthisis.

"We must characterize the instrumental treatment of laryngeal tuberculosis as very beneficial," say the authors, "and would not approach this disease without the double curette, until some better method is discovered (p. 87). Also, the extirpation of very swollen and infiltrated areas, although no more forcible method seemed to be indicated, not only caused an amelioration of existing symptoms and functional disturbances, but seemed directly commanded for humanity's sake.

"The double curette is better than the simple. The surface of the wound is covered with a scab either by the 75% lactic acid or the cautery, especially when it is not very large.

"The electrocautery—as it should be called instead of galvanocautery—deserved to be widely employed. That a reaction follows its use is conceded. To lessen it, the cautery should be used almost *white-hot* in order to attain the carbonizing of the tissue with the formation of a black or yellow scab. If it is insufficiently heated (red-hot) a white scab is formed, a necrosis, causing pain and dysphagia.

"Such scabs often require two to three days for sloughing, and are to be regarded as the result of insufficient incandescence. Care must be taken, therefore, to use the most intense heat, but to leave in contact the shortest possible time, repeating if necessary. If this is done, the reaction is minimal. It is over in a couple of days.

"Porcelain points are used for large surfaces and platinum for smaller. Grünwald's deep galvanocaustic puncture they consider an electrical overheating with metallic electrodes, since the point in its canal, even when the current is very strong, has a very faint glow or none at all, and heats the surrounding tissues so much that it leads to necrobiosis."

The results of the surgical treatment of Besold and Gidionsen were dependent on the general resistance of the patient, the severity of the local condition and the pulmonary disease, and finally on the correct and timely treatment. Of the 500 tubercular patients, 20 per cent were cured, and this lasted often eight to ten years.

As to the hemorrhage, which is supposed to be prevented by the galvanocautery, this is true only of the red-hot cautery, for the white-hot porcelain point does not prevent hemorrhage.

Why Grünwald was so afraid of artificial wounds could not be understood by the authors. On the contrary, they agree with other authors that this theoretic consideration was not borne out and that much oftener the artificial wounds very quickly were covered over and closed.

Friedrich (Kiel) in his last article opposes Grünwald's fear of injury to covering epithelium of the tubercular infiltrate.

Burger, in his last article on the importance of laryngeal tuberculosis, says: "Kronenberg's assumption that the results are dependent on chance does not seem to me permissible, because the most active, optimistic and restless champion of active treatment is always getting new results. And here, according to him, is the explanation of the distinct difference in the opinions of the writers. The surgical treatment demands not only a well developed technic, which, of course, is present in all the authors who have related their experiences, but it also demands a stock of patience, perseverance and faith. Whoever is scared off by a dozen disappointments and bad results and retires to the ranks of the nihilists, will have no correct judgment as to the possibilities of the surgical treatment. Entrance into this Holy Place is allowed only to believers. For the correct judgment of the value of this method, the hyperpessimistic, impatient and inexact therapist must be unconditionally excluded."

Everyone who has collected experiences extending over a long time in the surgical treatment of laryngeal phthisis, whether they be with cutting instruments or the cautery, will agree with Lermoyer, who has the following to say in regard to this method:

"In place of learning a new method, many find it easier to deny its correctness and results. Thus patients with laryngeal phthisis, who complain of fearful dysphagia, and are dying of hunger, are treated with gargles instead of procuring relief for them by curettement."

In agreements with Spiess and Krause, I have practically never seen a tubercular infection of a wound. In order to prevent the purulent infection of a wound, I use an alcoholic malachite-green solution of 1 to 2 per cent and can heartily recommend this after eight years' use. The astonishing disinfecting power of organic dyes was discovered by Koch in 1880 and investigated further by Behring. He showed that this remedy far exceeds the action of sublimate on anthrax.

In 1890 Stilling introduced methyl violet under the name of pyoktanin. This preparation has the power of checking suppurations and preventing them without causing any irritation of importance. Bresgen, by experiments extending over a long time, showed the advantages of this treatment, and I can substantiate everything he says. By the correct use of pyoktanin, we are not only able to favorably influence infectious and inflammatory conditions of the throat and tonsils and even cure them, but what is of more importance, are also able to avoid, after bloody or galvanocaustic operations in the nose, throat and larynx, an inflammatory reaction with all its sequelae, such as swelling, pain and suppuration.

I used pyoktanin for several years, until on the advice of Behring I gave the preference to malachite-green (Grübler). This drug has about three times the bactericidal power of pyoktanin. On some bacteria it has an almost specfic action. For example, it acts against anthrax and the comma bacillus about one hundred times as well as against the typhoid bacillus. According to Lingelsheim, streptococci cultures are killed in two hours by a proportion of 1 to 700.

For these reasons I use this drug for after-treatment, after the curettement and galvanocaustic treatment of tubercular ulcers and infiltrates.

The objections brought against this method of treatment are based on the following points: the malachite-green soils both physician and patient and sticks to linen and hands as green spots, which are hard to remove. In the larynx it causes a sensation of burning and dryness and has a taste, which to many is unbearable.

All these objections are of little importance when we consider its power of preventing inflammation, and must cause wonder that they are used as a basis for refusing its use.

If we understand it correctly, the objection is that malachite-green frequently stains the hands and laundry of the physician. The contraindication therefore is a purely personal objection to the loss of time in cleaning the spots.

Anyone who has used malachite-green for a long time and correctly, that is in fresh solution and proper dose, after operations on the mucous membrane of the upper air tract, will put into the background the inconveniences attending its use when he considers its very distinct advantages. By observing certain cautions, its bad features can be easily prevented. From my experiences I can give the following advice as to its use:

The malachite-green should be used only with a cotton-wound applicator, and only to the extent of a dark green coloration of the wound. Care must be taken to avoid an excess of the drug, by dipping the applicator in a vessel containing only enough fluid to be absorbed by the cotton. After the application the patient must neither cough nor spit. Only after some time may he take a drink of water. Many patients, however, on account of the bad taste, spit out the colored spittle at once, and stain their lips and chin. Cotton dipped in alcohol, will remove the stain. The patient must be warned not to spit into his handkerchief. In one's own interest, one should warn the patient not to cough, but if he does so anyhow, should be careful to get out of the way.

The well-known ink-spot eradicator (Weinstein's rods) removes the green color from the skin in a couple of seconds, as do ammonia and alcoholic potassium solutions.

When possible, the solutions should be made fresh, in small quantities, and kept from the light in dark bottles.

The first to use the galvanocautery in laryngeal phthisis was Voltolini. Discouraged by bad results he discarded this method.

Further experiments by Schaeffer, Pieniazek, Lublinski, Goughenheim and L. v. Schrötter gave relatively good results, but did not lead to its general use. A noteworthy article by S. Srebeny (*Medycyna,* 1893), who obtained cicatrization in almost every one of 13 cases, was not able to make it accepted, as the paper was published in Polish. In spite of my early bad results, observed in hospital practice, I have for 15 years used the galvanocautery, loop or point, in certain cases with advantage, especially in tumor-like infiltrations and granulation formations on the false vocal cords and posterior laryngeal wall, not only in tuberculosis, but also in scleroma and syphilitic granulation products. In such conditions they have given me the best of results.

In comparing the galvanocaustic with the purely surgical method, one point deserves special notice. Many writers claim that the use of the former in laryngeal phthisis demands a less degree of technic than the use of the cutting forceps. This view is eroneous. The correct use of the galvanocautery in the larynx requires a very exact technic and experience. The dosage of the heat is not so easy to learn as it is thought to be by many colleagues. Also, the form, length and thickness of the platinum point must correspond to the degree of destruction desired. All so-called galvanocautery knives must be discarded, to be replaced by double points, spiral points or small porcelain points. A complete severance of the infiltrated false cords with opening of the sinus and without burning or injury to the vocal cords demands a steady hand and a sure eye, and is technically as difficult as the use of Landgraf's double curette in this region. The same is true of all galvanocaustic operations on the vocal cords, especially their inferior surfaces. Here, every excess is a direct injury to the cords, from doing which I would give the strongest warning.

The possibility of hemorrhage after surgical intervention must not be overlooked.

Its frequency and danger seems to me to be exaggerated. Mermod is the one who insists on this most strongly. In more than 10,000 cases there were about 6 fatal and 15 severe hemorrhages. A fatal one two hours after curettement (from an angioma) was observed in my division of the St. Rochus Hospital by one of my assistants. I, myself, have seen eight severe hemorrhages in 1000 curettements, all of which could be checked.

M. Schmidt reports a case of fatal hemorrhage (the autopsy showed an angioma as the source) and a long, severe venous hemorrhage, which was checked by loosening the constricting clothing. Mermod saw a hemorrhage, from the results of which the patient died several days later. Two severe hemorrhages happened to my assistant, Dr. Wroblewski, after excision of the sclerotic false cords. I, too, have seen prolonged hemorrhages after operations on the false cords and the epiglottis, especially if the vein on the lingual surface was injured. I suspect that the number of copious hemorrhage is larger than is indicated by the literature, but I regard the wild fear of the many knife-shy colleagues as overdrawn and agree with Krause, who never saw a severe, unchecked hemorrhage in about 400 curettements, and who is disposed to refer it, when it does occur, to an improper technic. A few words here as to hemostasis. In parenchymatous hemorrhages, I use a 10 per cent cocain spray with an addition of adrenalin, followed by applications of small cotton tampons dipped in 5 per cent perhydrol. If it continues, a tampon dipped in a 20 per cent cocain solution is pressed against the bleeding area, and allowed to remain a half minute, followed by application of equal parts of lactic acid and liquor ferri sesquichloratum. In the case of small spurters (epiglottis) I apply, after thorough cocainizing of the bleeding area and pressing with cotton tampon dipped in perhydrol, the red-hot galvanocautery. Internally, secale cornutum is given. Also morphin for cough, ice collars, ice pills, absolute rest, speaking being forbidden.

In a case of dangerous hemorrhage from the false cords (scleroma laryngis) in a previously tracheotomized patient, I was compelled to use the double tampon by means of two tampons tied by ligatures. A double thread was passed by means of a Belocque's probe through the tracheal opening into the mouth and drawn outward. A small tampon was tied to the tracheal and a large gauze one to the oral end, both threads were drawn in opposite directions so that the smaller tampon was drawn through the tracheal opening below the vocal cords, while the larger was pressed against the introitus laryngis. After both had been fastened to the introduced tracheal canula, the hemorrhage was checked.

THE SURGICAL TREATMENT IS INDICATED.

(a) In tubercular tumors of the larynx.

(b) In circumscribed, chronic, tumor-like infiltrations of the posterior laryngeal wall, which show little tendency towards degeneration.

(c) In chronic ulcers, resting on an infiltrated base and surrounded by granulation products, which resist other methods of treatment.

(d) In unilateral disease of the larynx, even when the epiglottis, false cords and aryepiglottic folds are involved.

IT IS CONTRAINDICATED.

(a) In severe pulmonary phthisis combined with malnutrition and wasting.

(b) In diffuse miliary tuberculosis of the larynx, or of the larynx and pharynx.

(c) In all cachectic conditions.

(d) In severe laryngeal stenosis caused by inflammatory swelling of the affected parts (here the quickest possible tracheotomy is demanded).

(e) In frightened, nervous, irritative, distrustful, listless patients and those who frequently change doctors, but especially in persons whose general condition promises little for cure.

The surgical treatment demands from the physician devotion to the patient, patience and great persistence, as well as the most complete knowledge of the technic and an exact, sharp instrument. It should as far as possible not be performed on out-patients. The greatest attention must be used in the after-treatment, and the patients should be under observation for months and often years.

The operation is very slightly painful, if the cocain has been used correctly.

The relatives as well as the patient should be told beforehand that the trouble in swallowing cannot be cured at once by the operation, that it is frequently made worse for some days afterwards, and that the performance of the operation only in exceptional cases brings about a radical cure of the larynx. Furthermore, it would be well to state that the removal of the infiltration is rarely possible at one operation; that in spite of the operation, relapses may occur in the larynx; and that the physician can give no guarantee of the eventual cure.

After 20 years' experience I can confirm the conclusions stated in my address before the International Congress at Berlin:

"In combined phthisis of lungs and larynx, surgical intervention can cure localized disease of the larynx and restore its function. Nevertheless, relapses are not always prevented, whose further development on account of lesions in the lungs is not checked by the operation alone, without a corresponding general treatment, which latter must be always looked upon as the most important factor in the therapy."

I should like to add:

(1) The galvanocaustic treatment alone, or combined with surgical intervention, gives the best results in localized infiltrations and relatively good general condition.

(2) The fear of tubercular infection and severe hemorrhage is unfounded.

(3) Severe cases of laryngeal and pulmonary phthisis should be treated in sanatoria by specialists thoroughly trained in the technic.

As to the cure, I think only such cases should be considered cured where the larynx has regained its normal functions, i. e., vocal and deglutive power, where the general condition and nourishment leave nothing to be desired, where the pulmonary process has been checked, where cough and expectoration have disappeared, and where this favorable result has lasted at least one year without a relapse. An observation of several months is insufficient to place such an improvement in the category of "cured" (Besold and Gidionsen).

THE EXTERNAL OPERATIONS.

Here belong:

(1) Tracheotomy.
(2) Laryngofissure.
(3) Extirpation of the larynx.

Tracheotomy was first recommended by M. Schmidt, not only as an indicatio vitalis, but also as a curative measure, though Schmidt, in his book, says that since he has come to use the surgical treatment more and more and perhaps with better technic, he is finding less and less occasion to recommend tracheotomy. A further contribution to this question is furnished by Henrici. He recommends it for children, in healthy or slightly affected lungs, and relatively benign cases of laryngeal tuberculosis. Mermod is against tracheotomy as a curative measure, and employs it only in very severe laryngeal stenosis.

In hospital practice I have seen several times a tubercular infection of the tracheal wound in decrepit patients, with sputa filled with bacilli. The fault lay on the poor appointment of my division, insufficient ventilation and malnutrition of the patient. I have observed the local infection of the tracheal wound much less frequently in private practice.

Laryngofissure and Extirpation of the Larynx.—According to Grünwald, they are indicated in those processes or localizations whose cure seems impossible by the endolaryngeal method. Especially in perichondritic processes and their relapses, and also in very extreme disease we must think of external operations.

The first radical operation for laryngeal tuberculosis was performed in 1870, unintentionally, however, as the diagnosis was carcinoma. In the course of the following 35 years Grünwald found only 93 similar operations.

A very careful, statistical review of the histories, which unfortunately were not exact in many cases, gave the following:

67 per cent died soon after the operation.

8 per cent died from a short time to one year afterwards.

17 per cent were cured temporarily, without any report as to the subsequent course.

Grünwald designated the laryngofissure as an operation not dangerous in itself, so that in the future we need not take into consideration death from the operation, since it is connected with no danger to life. There is, however, the danger of tubercular infection of the wound through contact with pulmonary secretion containing turbercle bacilli, just as was observed in tracheotomy. This condition happens rarely. It can be prevented by operating only in healthy tissue, by performance of the operation only after an upper tracheotomy, and finally by an immediate closure of the wound. As to the influence of the laryngofissure on the general condition, an exacerbation was noticed in many cases, though this is not to be regarded as a rule.

Another disadvantage for the patient can arise through the formation of stenosis. This happens when two opposing surface are apposed and grow together. It can be prevented if it is possible to avoid long contract of the surface of the wounds.

The conclusions drawn from his statistics are expressed as follows:

Even in the initial stages of a lung affection, especially when the larynx is greatly involved, one should by all means refrain from extensive operations.

Extirpation of the Larynx.—The indications for this severe and often fatal operation are very limited. It demands the most perfect technic and the greatest care in the after-treatment, independent of the instinctive aversion of the patient to this severe operation, although favorable results have been obtained in this field. This is shown in the wonderful demonstrations by Prof. Gluck before this congress. In five patients operated for laryngeal carcinoma, lasting cure for years was obtained with restoration of a relatively loud and distinct voice. The patients breathe without a canula, swallow without difficulty, look all right, and are able to carry on their occupations.

In regard to the *phototherapy* attempted by Stillman and L. v. Schrötter, introduced by Sorgo, and tested by Kimwald, Jessen and Baer, I have no personal experience. In certain cases favorable results have been obtained, but their number is too small for positive conclusions and comparisons with the more energetic treatments.

Probably electric light will replace sunlight. The method is trying on the patient. Jessen allows the patient to be illuminated 5 minutes, and repeats this every hour. Still, I would advise methods which yield quicker results in cases where two weeks have passed without any distinct improvement.

I consider it a great service of Dreyfuss when in a discussion in our congress he insisted that physicians should send their laryngeal phthisis patients to us at a time when they are still curable. "I know," says the author, "that under certain circumstances distrust is caused by propagandistic acts. It will be called an unfair advertisement, a hunt for practice. These belittling thoughts must disappear and those who utter them be silenced when we declare to others that we are acting in the interest of a higher than personal motive, when we say to ourselves that we are doing nothing more than our right and duty, and when we take counsel for the combating of the plague of tuberculosis."

When I read these sentences of true humanity, I involuntarily thought of my home city, where, with a few exceptions, my attempts for years to introduce the surgical treatment has not only been fruitless but has been met by dead silence or discredited as injurious. I have never fawned on colleague or patient, never attempted to make everything alright and to swim with the current. The distrust of most colleagues for the surgical treatment is perhaps due to my own resignation and lack of patience in attempting to convince the principal opponents and their students. The present congress, which has given me the agreement of the most learned and skilled colleagues, will assist in removing the prejudices, help truth to victory, and bring to the unfortunate patient relief at least if not always a cure. As to the reproof that I am again bringing up a well-worn theme, I can answer with Goethe. "The truth must be continually repeated, for error is by the individual and by the masses."

Interest in this very important question has been again excited by the good results of Krieg, Mermod, Grünwald, Besold and Gidionsen, and lately of Friederich of Kiel, obtained by the use of the galvanocaustic, or the combined method (curette and galvanocaustic). The brilliant progress in laryngeal surgery, as laryngotomy and extirpation of the larynx, bring us in certain cases to the more radical methods.

I will conclude with the words of Moritz Schmidt in his preface to Besold and Gidionsen's work: "My cures have risen from 2 per cent to 20 per cent. We must continue to struggle in order that the results may be still better. The progress in the surgical treatment under the best aseptic precautions give us hope that this attempt will be successful. Therefore, to further fight along the entire line!"

XLII.

THE INFLUENCE OF ADRENALIN IN THE CAUSATION OF ARTERIO-SCLEROSIS.*

By F. E. Hopkins, M. D.,

SPRINGFIELD, MASS.

Since the publication of Josué's[1] experiments in 1903, considerable literature has appeared upon the subject of adrenalin as a cause of arterio-sclerosis. The marked uniformity in the results of the experiments reported during the four or five succeeding years, apparently established the case against adrenalin. In fact, judged by the influence upon the minds of many who read these reports or who saw references to them, the case was proven, since they hesitated to employ the agent or countenance its use. Later investigations have cast much doubt upon, if they have not entirely disproven the conclusions of these early observers; and that these later and more accurate reports may have as wide a dissemination as possible, I am led to ask your discussion of the subject, since your experience also with the use of adrenalin will be reassuring. Adrenalin has proven so useful and valuable an agent in our hands, both in allaying acute congestions and in affording a bloodless field for operation—not to mention other merits—that any suggestions of its responsibility in causing grave remote effects like arterio-sclerosis, arouses in one a feeling of resentment akin to that experienced when reflections are cast upon the integrity of an old and tried friend.

Having no original work to offer, this presentation shall be brief, yet that it may have somewhat of symmetry and historic sequence, reference will be made to some of the published observations, in the order of their appearance.

The attempt to produce arterio-sclerosis experimentally is not new, and a variety of methods have been employed to that end; such as the direct mechanical injury of the vessel walls, the intravenous injections of micro-organisms or their toxins,[2] the subcutaneous injections of nicotine, barium chloride, digitalin,[3] the administration of lead salts and tobacco, as well as the use of adrenalin. Since 1903 experiments have practically been confined to the latter, owing to the interest aroused by the report of Jores, of the Bonn Pathological Institute, who fed animals on adrenalin extract; and the reports already referred to, published by Josué the same year, who made use of adrenalin hypodermically, the injections being repeated daily for varying periods.

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*Read at the Thirtieth Annual Congress of the American Laryngological Association, held in Montreal, May, 1908.

Josué's method was adopted by the many experimenters in this country and Europe. Rabbits were used for the experiments, since the vessels of other animals were found resistant to degenerative action.

The reports of the findings of the various workers, including Miller,[2] Pearce and Stanton[4] in this country, Oscar Klotz[3] in Canada, Erb in Germany, Pic and Bonnamour in France, and many others, show a remarkable unanimity, and a quotation from one will serve for all. All agree that the smaller vessels are not involved, and that the media is the layer exhibiting the maximum degree of degeneration. Miles[5] says of the gross appearance of the lesions: "They were in general grayish-white in color, more or less opaque, circular to angular in outline, or sometimes appearing as short lines running lengthwise with the vessel, usually raised above the surface as the vessel was laid open, and varying in size from a pin-point to several millimeters in diameter. The larger areas were often somewhat uneven, both in elevation and outline. Those larger areas of calcification often showed tendencies to aneurismal dilations. Calcification could usually be recognized grossly in the larger lesions, and often in the smaller ones, by the more opaque white appearance of the lesion and by the gritty character when touched with a needle or scalpel. The lesions were more frequently found in the thoracic aorta, especially about the mouths of the branching vessels, often as an aggregation of pin-point areas, sometimes as larger plaques. They were frequently observed in the abdominal aorta and less frequently in the iliac arteries. Here also there was a greater tendency for them to appear about the mouths of the branching vessels, as in the thoracic region."

But he says in the same connection that the gross appearance of the lesions and their location is the same in the injected and in the non-injected animals. Also, that the result of no individual experiment can be predicted with certainty, no matter whether the dose administered be large or small, the duration of the treatment long or short, and he mentions the fact that the vascular system of normal rabbits has not been sufficiently investigated. In the same paper he publishes the statistics gathered by himself and Johnstone. Sixty-one rabbits received injections three times daily in amounts from ½ m. to 15 m. at a dose, and for varying periods. The total quantity administered to each varying from 3 m. to 1350 m. of a 1/1000 solution. The most extensive lesion was found in the rabbit receiving 5 m. doses, 150 m. in all, while many others receiving four to eight times that total showed absolutely no lesion. Of the sixty-one thus treated, seventeen, or 27.86 per cent, showed lesions of the large vessels on post-mortem examination.

They then examined fifty-eight "normal" rabbits, and twenty of these, or 34.48 per cent, were found to have arterial lesions of exactly the same character as the animals treated. These animals were from the same sources, and so far as possible kept under the same conditions as the rabbits experimented upon.

The foregoing report of Miles and Johnstone, together with the admitted fact that it is only in rabbits that vascular lesions have been found, and that it is questionable if the lesions in these animals are identical with arterio-sclerosis in man, together with the statement of all experimenters that they find it difficult if not impossible to produce lesions in young rabbits; that lesions are reported after only two injections (Braun reported by Miller[2]), and calcification as early as the fifth day after beginning treatment, that Klotz[3] found exactly the same results produced by barium chloride, and he states that the similarity is so striking that neither macroscopically nor microscopically can the lesions be distinguished from one another —all this variable and uncertain testimony, I say, throws doubt upon the assumption that adrenalin is the cause of the vascular lesions found after experimentation, and leads to the conclusion that most if not all the changes in the large vessels existed before the experiments were begun.

Practically this same conclusion is reached by Adler, who read an exhaustive paper upon the subject before the New York Academy of Medicine on March 19, 1908, a paper soon to appear in the *American Journal of the Medical Sciences.* On the same occasion Harlow Brooks read a paper to be published duly in the *Archives of Medicine,* and he reported a case, which goes farther toward proving the innocence of adrenalin under reasonable use, than can any amount of experimentation upon animals, or any theorizing, to prove the contrary.

Dr. Brooks' case was that of a woman thirty-five years old, who was under observation in a hospital for three years. She suffered from asthma to such a degree as to be totally disabled. No ordinary remedy afforded her relief. It was found, long after exhausting the usual list, that hypodermics of adrenalin overcame the spasm and brought relief for a longer or shorter interval. In view of the experimental evidence against adrenalin, its employment was sanctioned only as a last and deperate resort and because it was the only treatment which gave any relief. This patient received at times as much as 120 m. at a dose; and during the three years of its use forty-four one-ounce bottles of the usual 1/1000 solution were given hypodermically. She died after three years, but not from a cause attributable to the adrenalin; and the examination of her arteries revealed only the changes in vessel walls to be expected at her age. Nothing unusual was found macroscopically, but microscopically in the medial coat of the aorta were a few scattered points of degeneration, not sufficient to materially impair the strength of the vessel walls; and the patient might apparently have endured the hypodermics of adrenalin many years longer, had not her life been taken by other disease than that of her arteries.

In a word, then, the unreliability of animal experimentation to date, and the proven innocence of the large doses continuously administered in Dr. Brooks' case, give ample ground for reassurance in the reasonable use of adrenalin.

REFERENCES.

1. Quoted by Miller. American Journal Medical Sciences, April, 1907.
2. Miller, American Journal Medical Sciences, April, 1907.
3. Oskar Klotz. British Medical Journal, December 22, 1906.
4. Pearce and Stanton. Albany Medical Annals, February, 1906.
5. Miles and Johnstone. Journal American Medical Association, October 5, 1907.

ABSTRACTS FROM CURRENT OTOLOIC, RHINOLOGIC AND LARYNGOLOGIC LITERATURE.

I.—EAR.

Double Mastoiditis, Sigmoid Sinus and Jugular Thrombosis.

OPPENHEIMER, New York (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). A woman, aged 22, suffering from "la grippe," had a spontaneous perforation of the left mt., and four days later the right mt. was incised. Sudden high temperature was accompanied by vertigo, nystagmus, nausea and vomiting. Cerebration was slow. Tuning-fork tests showed diminution of higher tones. A streptococcus infection shown by culture. The left mastoid was opened and sinus exposed. Two days later the right mastoid was drained. Its sinus lay far forward and was wounded by the chisel, but the bleeding was easily controlled. Temperature 104° F., white blood count 19,000, polymorphonuclears 86%, five colonies of streptococci in each c. cm. of blood. The patient complained of chilliness, was restless, had headache and nausea.

On account of finding streptococci in the blood it was determined to open the sinuses. On the left side the sinus was uninvolved, on the right the wall was thickened and striated. On incision there was no free bleeding for some seconds, then it followed by a free gush. No thrombus was found, it being evidently washed out.

Temperature for the next three days was practically normal, yet blood cultures taken on the successive days showed increase in the number of colonies of streptococci from five to sixty to each c. cm.

Being preceded by a chill the temperature suddenly rose to 103.8° F. The right internal jugular vein was then exsected from clavicle to bulb. A series of enlarged glands were also removed. Above the facial the jugular vein was white, flat and densely adherent, while examination showed the presence of streptococci. From this time the patient made an uneventful recovery. A blood culture three days later was sterile. *Campbell.*

Case of Recurrent Keloid of Scalp and Lobule of Ear.

COCKS, New York (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). A man, aged 42, with multiple keloids. Four operations have been done and still they recur. Electrolysis and thiosinamin injections have been suggested for their removal.

Campbell.

Report of a Case of Mastoiditis, Complicated by Purulent Meningitis, Encephalitis, Phlebitis of Sigmoid Sinus, Jugular Bulb and Internal Jugular Vein. Operation. Recovery.

MCKERNON, New York (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). A man, aged 20, had an acute otitis media suppurativa with spontaneous rupture of the mt. At the end of five days pain recurred and the mt. was incised.

The head pains increased in severity and were accompanied by vertigo and vomiting. The pupils were irregular, the left being the smaller, pulse 100, temperature 104.4° F., respirations 24.

The right mt. bulged. The posterior-superior canal wall was collapsed. Pressure over the mastoid and tapping the skull caused him to cry out with pain. A smear of the ear discharge showed the diplococcus intercellularis meningitidis. The leucocyte count 11,600, with 94% polynuclears.

The entire mastoid was found infiltrated and was removed from the zygomatic ridge anteriorly to a point posteriorly which exposed the entire descending limb of the sigmoid sinus, as well as dura over a portion of the cerebellum. This removal exposed the entire floor of the middle fossa in this region. The dura bulged and was intensely red. On incising the dura several drachms of turbid fluid were evacuated, which contained the same diplococcus as that from the smear. For seven days following the operation the patient was unconscious, with all the symptoms of typical meningitis. During this time several lumbar punctures were made, and fluid withdrawn showed the same diplococcus as in the other examinations. The pupils were unequal throughout the entire seven days. On the eighth day consciousness returned and the ice-caps were removed. On the ninth day the patient was chilly. The temperature rapidly rose to 104.8° F. Tenderness developed along the anterior border of the sterno-mastoid and some enlarged glands could be felt.

The sinus was again exposed from a point one and three-quarter inches posterior to the knee above down to the bulb below. About the center of exposure the sinus was freely incised and a few drops of pus exuded, but there was no bleeding. The internal jugular was resected from skull to clavicle. Its lower two inches contained fluid blood, while above this was clot. The contents of the sinus were now evacuated and working backward towards the torcular a firm clot was found completely filling the lumen of the vessel; upon its removal a free hemorrhage was established. Sufficient bone was removed to permit the passage of a curette into the bulb and by little manipulation free hemorrhage was here established, the origin of the blood being, of course, from the petrosals. There was considerable sloughing of the posterior sinus wall extending as far back as the cerebellum, and this was removed by scissors. During the healing process there was a good deal of trouble with a hernia cerebelli until the entire exposed cerebellar cavity was mopped with a cotton-tipped probe dipped into a solution of silver nitrate 480 grains to the ounce. This occasioned an abundant slough, but there was no further protrusion. The neck wound healed kindly, and one year later the hearing on the affected side was normal.

Campbell.

A Case of Sinus Thrombosis, Following Removal of Granulation Tissue from the Middle Ear; Excision of the Internal Jugular Vein; Recovery.

DENCH, New York (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). Although not ordinarily supposed to be a dangerous operation, Macewen long ago called attention to the fact that removal of granulation tissue from the tympanic cavity might lead to serious intracranial involvement by opening up new avenues of infection through the freshly-cut surfaces.

The author's case is that of a young girl, who had a purulent discharge from the right ear since early childhood. Of late there had been gradually increasing pain, headache and sleeplessness. The meatus was almost completely filled with granulation tissue and the mastoid somewhat tender. Under general anaesthesia the granulations were removed and the temperature suddenly rose 5½° F. It fell to normal next morning, but the evening temperature was 106° F. As there was evidently an infective sinus thrombosis a radical mastoid operation was done. A firm clot was found in the sinus, which was removed by curette, but as no bleeding came from the region of the bulb, the internal jugular vein was excised. Microscopical examination of the clot showed a streptococcus infection.

Ten days after the radical operation the cavity was lined with a Thiersch graft and the posterior wound closed.

Campbell.

Report of Cases of Mastoiditis in the Aged.

ALDERTON, Brooklyn (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4), reports seven consecutive and unselected cases in five of which there was no perforation of the mt. or discharge, while in two there was perforation with granulations and purulent discharge.

In the majority there was no marked systemic disturbance; headache, neuralgias, sleeplessness and tinnitus being the symptoms mostly complained of. The inflammation was low grade and long duration, attributable to the lessened tendency to reactive inflammations, which pertains to advancing years. This was noted in the resistance of the coverings of the brain and its sinuses to the septic foci in direct juxtaposition.

Campbell.

Do Not Remove the Thrombus.

VOSS, Riga (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). In cases of suspected sinus thrombosis from the moment one begins exposing the sinus, bear in mind that unnecessary manipulation is highly injurious, because dislodging portions of the thrombus into the general circulation will cause metastases.

Do not palpate. Do not remove the thrombus. Make a diagnosis by the eye and aspirating syringe. On aspirating the sinus contents one can often obtain a half gram of dark fluid, which when squirted out on a white background is recognized as brownish-red and not venous colored. On diagnosis being made the jugular is ligated and divided and the sinus is freely exposed and incised, going 1—1½ cm. into healthy parts. The entire outer wall of the sinus is cut away with scissors so that the thrombus is exposed, but it is allowed to remain in situ. Free drainage of the infectious material is now assured, and the thrombus is left to its fate. *Campbell.*

Eustachian Catarrh with Hyperinflation; Report of a Case.

BRAISLIN, Brooklyn (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). A young man experienced a sensation of fullness in the ears, and slight dullness of hearing with each act of swallowing or on talking. There was likewise a sensation when speaking of talking in a resonant space. These symptoms have persisted since an attack of rhinitis one year ago.

While talking he "sniffs," as this gives temporary relief, but only until the next act of swallowing, speaking or chewing. Posterior rhinoscopy shows hypersecretion at the mouths of the Eustachian tubes. Valsalva's experiment exaggerated the symptoms. Removal of the posterior ends of the inferior turbinates overcame the difficulty. *Campbell.*

A New Instrument for the Treatment of Diseases of the Ear.

FOWLER, New York (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). The apparatus consists of a glass bell, which incloses the ear and fits closely to the tissues round about. From the center of the top of the bell a nipple projects for attachment to the fountain syringe tube, while projecting inward from the nipple is a nozzle to which soft rubber tubing is attached for passage into the external auditory meatus.

On the circumference of the bell is placed an outlet nipple to be connected with rubber tubing for drainage into a low receptacle. This outlet tubing exerts suction action. The fountain syringe fastened at a point two feet above the head will give the ordinary safe nozzle velocity. *Campbell.*

Otitic Sinus Thrombosis and Pyemia.

ALEXANDER, Vienna (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). Otitic sinus thrombosis is caused by infection of the sinus contents by continuity or metastasis from middle-ear suppuration. The infection may be imparted to the entire body from the diseased sinus and this is the time when the pyemia and the bacteremia begin. Usually bacteremia in a few days results in death, whereas pyemia leads to metastatic, purulent inflammations.

According to the position of the metastasis we distinguish four kinds of otitic pyemia, viz.: intracranial, thoracic, abdominal, articular and in the muscles; of these intracranial metastases is by far the most frequent.

The symptoms of sinus thrombosis are substantially those of pyemia—intermittent fever, chill and spontaneous pain. There is usually a sudden change in the amount of middle-ear secretion; it either stops or greatly increases and flows continuously.

The operative indications are:

1. Removal of the focus of infection.
2. To confine the pus to the ear, thus preventing the formation of metastasis.
3. The establishment of good drainage.

One always begins with ligation of the jugularis about its middle third; when it contains a thrombus it is cut through and the upper end sutured to the upper angle of the neck wound. The vein below this is laid open until well beyond the thrombus. This ligation is done so that in immediate operation on the ear one is not afraid of dislodging particles of thrombi during the jarring produced by chiselling and there is no danger of air aspiration in opening the veins and sinuses. When the sinus region is sufficiently exposed in the radical operation, the sinus sigmoideus is incised 2 mm. in length with a scalpel. If there is no hemorrhage the sinus is cut longitudinally and the thrombus first removed from the peripheral end, and then from the central end and the bulb. Aspiration of sinus contents does not furnish a reliable result, for with a parietal thrombus fluid blood can be aspirated. In such a case after incising the sinus with a scalpel, the blood is apt to flow with diminished force. We cover the incision for a few minutes with sterile gauze and then frequently find the thrombus in the gauze, it being loosened and carried there by the flow of blood. In all cases every attainable thrombus must be removed. *Campbell.*

The Present Status of the Radical Mastoid Operation for the Cure of Chronic Purulent Otitis Media.

WENDELL PHILLIPS, New York (*Medical Record*, Oct. 10, 1908), states that permanent cessation of the purulent discharge does not take place in more than about 65 per cent of the cases operated upon, even in the hands of the most skillful operators. Many of the remaining have but intermittent discharge or are victims of reinfection, others throw off cholesteatomatous masses, and still others are victims of further extension of the necrotic process. These statistics show that the end results are less rosy than the first enthusiasm seemed to imply.

The radical operation is indicated: (1) When a permanent cessation of the purulent process has not been effected by prolonged local intratympanic treatment, combined if necessary with such minor operations as removal of granulations, enlarging perforation, etc. (2) When the cure has not been effected by the removal of necrosed ossicles and the curettage of the middle ear. (3) When acute symptoms of mastoiditis are present. (4) When a sudden cessation of the pus discharge produces vertigo, pain, or other unusual symptoms. (5) The appearance of facial paralysis during the course of chronic purulent otitis media. (6) Attacks of vertigo, indicating that the necrotic process involves the labyrinth. (7) In all cases where intracranial or lateral sinus involvement has already appeared. (8) Where there are positive symptoms of cholesteatoma in the mastoid antrum. (9) Where there are fistulous openings in the cortex of the mastoid process or in the osseous canal wall. (10) Whenever extreme depression or other symptoms of disturbed mentality accompany the disease.

The operation is contraindicated: (1) When the purulent process is tuberculous and accompanied by advanced general tuberculosis. (2) In advanced pernicious anemia or albuminuria and in cachectic diabetes. (3) It is usually contraindicated in young children. (4) In all cases where the disease is confined to the ossicles and tympanic cavity. (5) In adults who have scanty otorrhea without odor, with improper opening of the drum membrane, behind which are retained masses of secretion. (6) In all cases where it is possible to effect a cure by any of the other methods.

He advises against the radical operation in young children for the following reasons: (1) The disease is rarely of sufficient extent to require it. (2) The disease usually yields to local treatment, providing diseased tonsils and adenoids are removed. (3) By establishing free drainage, through a large perforation, with well maintained local treatment, a cure usually results. (4) The natural recuperative powers of childhood may bring about a cessation of the discharge, whereas there is the danger of partially or wholly destroying the function of hearing, with the train of misfortunes which follow.

Richards.—

Chronic Otitis Media Purulenta.

Alfred Wiener, New York (*Medical Record*, Nov. 7, 1908). Proper drainage and conservative measures, followed by ossiculectomy, should precede radical treatment. The author's experience has been that in those cases in which conservative measures are attempted with success a great deal more is to be expected and can be promised than when the radical operation is performed.

When this is performed, a cure will be established in many cases, while in others, in spite of the operation, the hoped-for cure will not be brought about. *Richards.*

Cerebral Abscess.

Emil Amberg, Detroit, Mich. (*Journal A. M. A.*, Aug. 22, 1908). Patient was a girl of about 12 years of age, whose ears began discharging soon after birth. Diagnosis of sinus thrombosis was proved by operation. Thrombosis removed. Child did not do well. Second operation nine days later. Internal jugular vein first tied, then the middle cranial fossa opened and about two drams of yellowish-green pus evacuated. Death eight days later.

The deep-seated brain abscess was probably old, the child having been subject to occasional violent attacks of pain in the head, intensified when she blew her nose. *Richards.*

Nasal Analgesia as a Prognostic Symptom in Dry Catarrhal Deafness.

Dunbar Roy, Atlanta, Ga. (*Journal A. M. A.*, Aug. 29, 1908), has found that many of his patients consulting him for progressive deafness presented at the same time a condition of manifest nasal analgesia, by which he means manifest insensitiveness of the nasal mucosa, allowing one to accomplish all kinds of instrumental manipulations in these cavities without discomfort to the patient and without the use of any local anesthetic. This analgesia is also present in the ear itself. He has not yet determined whether the same condition that exists in the cases of progressive deafness is also present in the sensitive nerve terminals, but is convinced that the same conditions are present in the mucous membrane in the nose, nasopharynx, Eustachian tube and middle ear; and the more advanced these changes in the mucosa and nerve terminals are, the less favorable is the prognosis for the improvement of hearing. These same degenerative changes also finally take place in the auditory nerve itself, as is evidenced by the tuning-fork test and the histopathologic investigations of Moos, Politzer, Gruber and others. *Richards.*

A Report of a Case of Brain Abscess Associated with Chronic Purulent Otitis Media, with Special Reference to the Operative Technic Followed.

John Randolph Page, New York (*American Journal of Surgery,* September, 1908). Patient was a child of six years and first complained of headache, then vomiting and convulsions, first supposed to be due to intestinal disturbance, but later thought to be due to the ear. The right ear discharged, and 24 hours after admission to the hospital the veins in the fundus of the right eye were more swollen and tortuous than in the left and the edges of the disk more blurred. The mastoid and squamous portions of the bone being exposed, an opening into the cranium, large enough to admit of using a rongeur, was made with a gouge, and the tegmen antri and tympani were removed. They were softened by necrosis, but there was no perforation. The antrum and middle ear were filled with necrotic material and foul pus. The bone covering the outer aspect of the temporosphenoidal lobe was removed upward for little over an inch, forward for an inch and backward for about three-quarters of an inch. There were no granulations or lymph on the dura or any tract of infection discovered. Its tension was plus one or over. A narrow straight bistoury was introduced into the brain, upward just above the tympanum and then over the antrum, with negative result. Three rows of punctures, three or four punctures in each row, were then made, one-third inch over each other, but no pus was found. More bone was then removed anteriorly for one-quarter of an inch or more and three more punctures made the same way. Still no pus. Bone was then removed posteriorly, and after two unsuccessful punctures the bistoury was introduced almost straight in, just above the angle made by the entrance of the petrosal into the lateral sinus, and on slowly removing the knife there was a flow of pus. A half inch crucial incision was then made in the dura at this point, followed by a discharge of pus. In the absence of an encephaloscope, a large-sized Gruber ear speculum with a gauze obdurator was carefully introduced just within the incision, the obdurator removed, and the pus that appeared in the speculum was wiped out with absorbent cotton. This was done a number of times, until about half an ounce of pus was removed. The opening in the dura was held open with gauze packing, a small wick of gauze having been carried into the opening in the brain as far as could be seen and left there. The anterior part of the wound was sutured. The dressing was changed twice a day for four days, but as the abscess seemed to drain well, the wick was not changed until the second day, when it was carefully removed and replaced, being changed every day but not removed entirely until three weeks after the operation. A small hernia about the size of a hazel nut followed the operation, but at the end of four weeks it was completely closed off and the whole area presented a uniformly granulating surface.

The author does not believe in introducing the finger into the brain, and regards tubes and rubber tissue more unsatisfactory for drainage than gauze. *Richards.*

Tuberculosis of the Ear.

W. Sohier Bryant, New York (*Medical Record,* September 26, 1908). The middle ear inflammation presents a peculiar picture. One sees an indolent, serous or sero-purulent, more rarely a purely suppurative, inflammation, melting away of the drum membrane, multiple or kidney-shaped perforations, exposure of the manubrium mallei, etc. Continuation of the ulcerative process in the face of ordinary treatment is pathognomonic of tuberculosis, especially alternating increase and decrease of the activity of the process independent of other obvious causes. Tympanic tuberculosis is a very early sign of a more general infection and also gives an accurate indication of the course of a concomitant pulmonary tuberculosis. The author has found the pyoctanin dry treatment to be the most efficacious. *Richards.*

Report of Two Interesting Cases of Mastoiditis.

J. H. CRAWFORD, Atlanta, Ga. (*American Journal of Surgery,* September, 1908). Two cases of primary mastoiditis are reported. The first was an acute case of traumatic origin, as a result of injury from a piece of copper ore. The second occurred in an infant of eleven months, and was apparently primary mastoiditis, as there was no discharge from the ear and the child had given no evidence of being in pain. The tip cells were large and broken down. Operation was done in both cases, with rapid and uneventful recovery.

Richards.

The Myth and the Mystery of "Meniere's Disease."

GEORGE M. GOULD, Ithaca, N. Y. (*Medical Record,* Oct. 31, 1908). At the end of a fifteen-column article, reviewing very much at length much of the past literature of Meniere's trouble, the author reaches the following conclusions:

"Meniere's Disease" is simply "migraine," or sick headache, with many symptoms ignored and with three of the secondary or incidental effects of migraine over-emphasized:

(a) Vertigo.

(b) Tinnitus (a) of a purely functional neurotic nature, a reflex-ocular neurosis to the center or to the organ of hearing; (b) of traumatic origin, caused by vomiting.

(c) Partial or complete deafness, due to injuries in vomiting.

Migraine is one of the evil effects, the most common and the most important of eyestrain, always preventable, almost always curable by scientific spectacles, because in almost one hundred per cent of cases it is due to ametropia.

Richards.

Acute Otitis Interna, with Report of a Case.

EDGAR R. STILLMAN, Troy, N. Y. (*Medical Record*, October 3, 1908). References to this condition during the past twenty-five years are few and incomplete, as the patients always recover. A perfectly well child is seized suddenly with vomiting, headache, occasionally with earache, high fever and sometimes with a convulsion. In three to eight days the force of the disease has spent itself, but the child is completely deaf. It gets well completely with the exception of a staggering gait, but remains deaf. The keynote to the diagnosis is early and complete deafness, with rapid recovery in a child whose symptoms indicate some acute intracranial inflammation. A case is reported in a child of seven years who was sent home from school because of headache, pain over the right eye and feverishness, following slight delirium the previous night. Objective examination was negative, and there was nothing to cause suspicion of any trouble with the ears or of the vital processes. Temperature 103°. A restless night followed and there was prostration the following morning, the child being only aroused by shaking and then would glance around in a strange sort of way and close his eyes again. He would not answer questions and seemed not to comprehend their meaning. Fever had subsided somewhat and he slept all the time, except when roused for medicine or nourishment. Next day it was discovered that the boy was totally deaf. There was no pain or soreness about the ears. Examination with the tuning fork showed absolute loss of both aerial and bone conduction. No evidence of any other cranial nerve involvement. He was still a little feverish, but his general condition seemed to be somewhat improved. He could recognize objects and could read as well as usual. Next day he played in bed, but was still absolutely deaf. At the end of the week he staggered on attempting to walk and had a gait resembling cerebellar ataxia or that of a drunken man. Twenty-five days later the child was apparently as well as ever, but totally deaf.

The etiology of this disease is very obscure, and diagnosis is only possible after the characteristic symptom of total deafness has developed. Prognosis is invariably favorable as to life. *Richards.*

Ear Symptoms of Cardiovascular Disease, with Special Reference to Low Pressure Cases.

Louis F. Bishop, New York City (*Journal A. M. A.*, November 7, 1908), thinks that there are certain cases of tinnitus aurium which are a local manifestation in the general circulatory disorder, which may have its foundation in arterial sclerosis, but which is often in a large measure functional so far as the blood vessel disorder is concerned. It is found in connection with high blood pressure and also with low, and while in both instances the symptom is relieved by measures which regulate the circulation, the most striking results of treatment are to be obtained in cases of low arterial tension.

He thinks that otologists should make observations of blood pressure, so as to detect gross departure from the normal, and he has devised a method by which this can be done in a very few seconds.

In the way of treatment the most important element is systematic exercise. Iron and arsenic are of value as internal medicines. *Richards.*

Distinct Pulsations in a Non-Perforated Membrana Tympani.

MAX FIEDLER (*Archiv. f. Ohrenheilk.*, Bd. 76, Heft 3 u. 4, 1908, p. 256) reports a case of a woman 48 years old, who for two years had had subjective tinnitus in the left ear. On the left side a marked central retraction of the drum was seen and in the anterior upper quadrant a pulsation synchronous with the radial pulse, producing at each beat two reflex lights. In the right ear a similar pulsation could be seen, but it was placed more centrally and somewhat deeper. Contractions of the arteries of the neck caused disappearance of the phenomena. It remained unaltered after catheterization and the use of the Siegle's speculum.

Wood.

The Employment of Hot Air in Otology.

FERRAN (*Revue Hebdomadaire de Laryngologie, D'Otologie*, August 8th, 1908) has used hot air in the treatment of diseases of the ear for the last two and a half years. His results have been so encouraging that he feels it is a valuable means of treatment in many cases. A chief objection in the past to the use of hot air has been the lack of a simple apparatus. This, the author states, he has overcome by the use of a simple Politzer bag, prolonged by a muff of aluminum containing an electric rhostat. With this in from four to five minutes a temperature of 70 to 80 degrees C. to be obtained. The apparatus permits the use of the catheter as well as the

Politzer bag. (The author gives credit to America for the idea of the apparatus.) The abstractor has not seen this particular instrument. He has had, however, entire satisfaction with that made by Wappler Brothers, New York, with which the same amount of heat can be obtained in a much shorter time. Ferran is in the habit of giving two treatments a week, occasionally, however, making them daily. He has used it in 217 cases. While in cases of pure otosclerosis, he admits failure, as far as cure of the disease is concerned, he has been able to give relief to many of the accompanying symptoms. In cases of chronic middle ear disease with more or less stenosis of the tube, he has gotten his best results. Here, as shown in several of the reports which he gives, he has succeeded in not merly relieving the tinnitus, but also improving the hearing. He has noticed a rapid disappearance of the ear symptoms which have followed an attack of acute rhinitis, especially if it was of a grippe variety. In acute inflammation of the middle ear with perforation, benefit has been noticed from the use of hot air, probably through its effect upon the Eustachian tube, and in certain forms of middle ear suppuration he has also obtained benefit. Here he has used the hot air in the middle ear directly through the auditory canal. The beneficial results obtained are to be explained in his opinion to the effect on the mucous membrane of the tube of the naso-pharynx similar to that produced by cocain or adrenalin.

Harris.

Case of Double-sided Necrosis of the Inner Organ of Hearing, With Fatal Hemorrhage From an Aneurism of the Internal Carotid.

C. E. BENJAMINS (*Archiv. f. Ohrenh.*, 3 und 4 Heft, Bd. 76, 1908, p. 240) reports a case in which arsenic had been applied to the external auditory meatus for ear trouble. Necrosis of both petrous bones followed, and associated with this was paralysis of both facial nerves. A radical operation was performed, under chloroform, on both sides, on July 21st. The patient for a while did well after the operation, but on August 25th and afterwards at irregular intervals, severe hemorrhage occurred from the right ear. A diagnosis was made of venous hemorrhage, probably from the jugular bulb. As the patient was very anemic, immediate operation was decided upon, and the transverse sinus was packed and the jugular vein tied. Nevertheless, on the night following the operation the patient died from sudden hemorrhage. The post mortem examination showed extensive necrosis of the petrous pyramid, aneurism of the internal carotid artery with thrombosis and rupture, also very poor development of the right transverse sinus and enormous dilatation of the left.

Benjamins says that in cases where hemorrhage of the carotid is diagnosed the best treatment is packing of the middle ear after a radical operation, combined with tying of the common carotid artery. Simple ligation of the carotid does not prevent a return flow from the circle of Willis, and it is difficult by packing alone to absolutely check a carotid hemorrhage. It must be remembered, however, that ligation of the common carotid is often accompanied by hemiplegia and other brain disturbance, ten per cent being fatal.

Wood.

Histology of Suppurative Processes in the Labyrinth.

E. Ruttin (*Beiträge z. Anat. Phys. Path. u. Therap. d. Ohres. Nas. u. Halses,* Bd. 1, Heft 5 u. 6, 1908, p. 388) examined 3 cases of suppuration of the labyrinth, probably tuberculous in nature. In all three cases the pus entered the perilymphatic space by way of the posterior wall of the promontory, breaking into the scala vestibuli. From a study of these cases, and a rather extensive review of the literature of the subject, he deduces the following conclusions:

1. Circumscribed chronic suppuration of the perilymphatic space of the vestibule produces by gravity an associated disease of the perilymphatic space of the scala vestibuli, involving the lower half of the first turn.
2. This is followed by a peculiar ectasis of the ductus cochlearis, giving to it a pyramidal or vault-like shape. If a rupture takes place into the endolymph, the point of the rupture is always placed in the dependent portions of the membranous labyrinth (sacculus, ductus reuniens, cecum vestibularis or the vestibular portion of the ductus cochlearis).
3. A chronic circumscribed suppuration of the first turn, on account of gravity, may be sharply localized in the lower half of the turn.
4. A chronic circumscribed suppuration of the scala tym-

pani, produced by a rupture through the round window usually remains limited to the same, if the basal membrane is thick. If a rupture takes place its location is in the basal membrane.

5. A very important fact from the clinical side is the difference in importance of the location of the seat of rupture into the labyrinth. If the suppuration breaks through into the vestibule the mechanical relations bring about an associated disease of the cochlea. Suppuration which finds its way to the labyrinth through the round window, likewise for mechanical reasons generally remains limited to the lower half of the first turn of the cochlea. Therefore, the prognosis for the function of the labyrinth is much less favorable if the rupture takes place into the vestibule, because every time in these cases the entire labyrinth becomes diseased; while on the other hand the favorable prognosis of rupture through the round window is because of the limitation of the infection through a small portion of the cochlea.

According to many authors, suppuration following rupture through the semi-circular canals is still more favorable.

Wood.

Concerning the Clinical Aspect of Labyrinth Suppuration.

FRANZ NUERNBERG (*Archiv. fur Ohrenheilkunde,* 1908, 76 Band, 3. u. 4. Heft, p. 139, 230) reports 26 cases of secondary affection of the labyrinth occurring in 3,695 cases of suppuration of the middle ear. Each case is reported in considerable detail. In conclusion he says that:

1. The large majority of fistulae of the semi-circular canals arise through erosion of the semi-circular canal from the outside and are mostly circumscribed lesions.

2. It is possible to have a primary circumscribed suppuration of the labyrinth with the retention of hearing, and, therefore, the presence of hearing does not exclude a suppuration of the labyrinth, especially if the suppuration is limited to the vestibular apparatus.

3. It is not possible to distinguish accurately between the irritation and paralytic phenomena of the vestibular apparatus, as suggested by von Stein and Hinsberg.

4. Suppuration of the labyrinth is not always accompanied by disturbances of equilibrium, and in relation to this it is

worthy of note that a negative result of Hinsberg's modification of Stein's test is not a positive indication that the labyrinth is intact.

5. A positive Barany's test of the vestibular apparatus proves that the vestibule is functionally active, but it must be remembered also that when this organ is markedly involved, the Barany's test may still be positive though the reaction is a very slight one.

6. If we exclude the cochlea, the disease of the labyrinth can only be diagnosed when the hearing becomes rapidly diminished at the beginning of the disease, or during our observation, and finally sinks to complete deafness.

7. The vestibular apparatus is much more resistant to the inflammatory process than is the cochlea.

8. Fistulae of the semi-circular canals are not always signs of an active inflammation of the labyrinth; quite frequently they are the residuum of a healed labyrinthitis. In such cases, symptoms of labyrinth involvement may be induced by pressure of cholesteatoma, and disappear when these masses are removed. *Wood.*

Histologic Studies Concerning the Changes in the Tympanic Wall of the Labyrinth, Following the Radical Operation for Cure of Middle Ear Suppuration.

Erich Ruttin (*Arch. f. Ohrenh.*, 1 u. 2 Heft, Bd. 76, 1908, p. 81). Ruttin's research was based upon the histologic findings in 25 cases of chronic middle ear suppuration, on all of whom the radical operation had been performed. With the exception of 4 cases they all died from intracranial complications. The most frequent change in the epithelium is the formation of cysts which in the larger majority of cases result from the displacement of tissue by the operation. These cysts the author divides into four types. The first type, which has already been described by Politzer and others, is due to the projection of folds of inflamed mucous membrane which coming in contact with each other, form a sort of enclusion cysts, lined on all sides with mucous membrane. They may be formed on any part of the tympanic mucosa except that covering the membrana tympani. While these cysts may occur in unoperated cases of otitis media they are much more common after the radical operation. The more thorough the curetment, the less liable is the formation of this variety of cysts. The second variety of cyst results from failure to remove the mucosa from the depression of the oval window. They are generally three-cornered in shape, with the point of the triangle generally turned towards the insertion border of the stapes. Although these cysts occur in non-operative cases of chronic suppuration, they are more frequently seen after operation on account of the curetment pushing a tag of mucous membrane over the edge of the depression of the oval window. This second variety of cyst may be lined either with a cylindrical epithelium containing goblet cells, or with a metoplastic epithelium of squamous cells. Frequently there is a third variety of cyst formed in the round window, due to the over-growth of the surrounding mucous membrane. This over-growth may take on the form of a pedunculated polyp, or a solid tumor of granulation tissue, which grows over the round window, and because of its uneven surface becomes irregularly attached, the spaces between the attachment forming occlusion cysts. The fourth variety of the cyst-like formation is due to a rapidly growing epithelium spreading over a depression which is already filled with pus.

Beside the cyst formation, there is frequently found some pathologic change in the foot-plate of the stapes, in the annular ligament, in the membrane of the round window, in the base of the promontory, and finally in the cellular spaces of the lateral pyramidal wall. The foot-plate of the stapes may be dislocated either towards the vestibule or towards the tympanic cavity or it may be completely or partially absent. If the infection passes through the oval window a circumscribed inflammation is very apt to become changed into a diffuse infection of the labyrinth. The round window acts much less frequently as a port of entrance for infection to the labyrinth than does the oval window. In ordinary suppuration of the middle ear, inflammation of the labyrinth takes place almost always through these two. If cholesteatoma or tuberculosis is present the penetration may take place through the promontory or the lateral wall of the semi-circular canals.

Infection of the supra and intralabyrinth cells is generally a chronic suppurative infiltration, with granulation tissues growing into the medullary tissue of the bone. This can under certain conditions lead to the sequestration of the entire labyrinth. *Wood.*

Intracranial Disease of Otitic Origin.

H. E. Kanasugi (*Monatsschrift f. Ohrenheilkunde*, etc., vol. 42, Heft 5, p. 238) reviews the literature on the subject and reports the result of his own postmortem examinations in 13,400 necropsies. Intracranial disease of otitic origin was found 42 times.

1. Variety of ear diseases. Otitis media purulenta acuta, 6 cases; Otitis media purulenta chronica, 30 cases; Otitis media cholesteatomatosa, 2 cases; Otitis media purulenta chronica and Labyrinthitis purulenta, 4 cases.

2. Variety of lesion in brain. Cerebral abscess, 14 cases; Cerebellar abscess, 6 cases; Cerebral and cerebellar abscess, 2 cases; Cerebral abscess and meningitis, 5 cases; Cerebral abscess and sinus disease, 4 cases; Sinus disease, 5 cases; Meningitis, 4 cases; Cerebellar abcess and meningitis, 1 case; Sinus disease and meningitis, 1 case.

3. Sex. In the 42 cases, 31 were men and 11 women.

4. Age. Greatest number occurred between 10 and 30 years. Seventeen of the 42 cases occurred between 10 and 20 years.

Twenty-seven of the forty-two cases occurred on the right side and fifteen on the left side. *Wood.*

Facio-Hypoglossal Anastomosis.

George F. Cott, Buffalo, N. Y. (*The Journal of the A. M. A.*, August 8, 1908). This article gives the detailed technic of the operation, with numerous drawings, and should be read in its entirety. *Richards.*

Thrombosis of the Lateral Sinus.

Philip Hammond, Boston (*Boston Medical and Surgical Journal*, Sept. 24, 1908). Five cases of thrombosis of the lateral sinus, all operated on successfully, are reported.

Richards.

The Cochlea and Its Sensitiveness to Tone Impressions.

W. Sohier Bryant (*Archiv. f. Ohrenh.*, 1 u. 2 Heft, Bd. 76, 1908, p. 44) from a study of cases which he has collected from literature, does not seem to arrive at any definite facts concerning the functional tests of the internal ear. He says in conclusion, that the reported protocole of functional examinations and the following post-mortem findings is lacking in sufficient detail to arrive at definite conclusions, but it seems to indicate that the different parts of the cochlea are not limited in their sensitiveness to tone, and that each part exercises its function for all tones without distinction.

Wood.

II.—NOSE.

The Surgical Treatment of Orbital Complications in Diseases of the Nasal Accessory Sinuses.

Arnold Knapp, New York (*The Journal of the A. M. A.*, July 25, 1908). The orbital complication referred to is the subperiosteal abscess. In the acute cases the local symptoms are those of an acute abscess with cellulitis. In the chronic variety there is more or less defect in the bony orbital wall of the frontal and ethmoidal sinuses, and with the orbital periosteum a large abscess cavity is formed, causing marked exophthalmos. In the chronic cases of exophthalmos and in other cases with acute perforation which do not improve on the restoration of intranasal drainage, the method of operating is, first to remove the anterior half of the middle turbinate a day or two before the external operation; second, under morphin-ether narcosis, make an external incision along the upper orbital border midway between the eyebrow and the bony orbital margin, then down along the inner wall and side of the nose to the floor of the orbit. Divide the periosteum at the orbital margin above and in line with the cutaneous incision along the nose. Retract the periosteum with a rather sharp elevator, and gently detach the lachrymal sac after the firm adhesion to the orbital margin is separated. Free access is thus given to the roof and the inner wall of the orbit. The pulley of the superior oblique is carefully detached from the trochlear fossa. It is important not to disturb the relation between the tendinous ring through which the tendon passes and the periosteum to which it is adherent. The entire floor of the frontal sinus is then easily removed with the chisel, the diseased mucous membrane curetted, and the bony septa in the frontal sinus carefully eradicated. A portion of the orbital margin can be removed without causing deformity if the normal curve be preserved. Next resect the nasal process of the superior maxillary, the lachrymal bone and the ethmoidal os planum, so as to give broad access to the ethmoidal labyrinth, which is completely removed with the çurette. The anterior ethmoidal foramen is a constant landmark, indicates the base of the skull, and as one proceeds back, shows that the ethmoid becomes broader laterally. The remaining part of the middle turbinate may now be removed and the sphenoidal cavity entered if necessary. As broad an opening into the nose as possible is made in addition to removing all disease, to insure proper drainage. Should the frontal sinus extend unusually high, and the upper limit cannot be curetted from below, the cutaneous flap with the eyebrow is forcibly retracted upward, and a window cut in the anterior bony wall similar to the Kuhnt and Killian methods, leaving a broad bony supraorbital margin covered with periosteum, but only a sufficient amount of the anterior bony wall is removed to treat properly the upper parts of the cavity under direct inspection. Marked subsequent sinking in of the forehead can thus be prevented. The cutaneous wound is not sutured, the soft parts approximate of themselves, and a single wick of gauze is passed into the frontal sinus. Slight packing is introduced if bleeding demands it, which is removed after 24 hours. The patient occupies a partly upright position in bed. The external wound closes primarily. The small opening for the drain is left at the inner orbital angle for from seven to ten days. The nasal cavities are left undisturbed. In some cases diplopia is noticed for a few days, but always disappears. *Richards.*

The Ethmoid Cells at Birth and Their Development During Fetal Life.

E. J. CURRAN (*Boston Medical and Surgical Journal,* Oct. 29, 1908). This paper gives the results of some anatomic studies and the development of the ethmoid, and is based on eighteen dissections. All the nasal sinuses seem to be formed by outgrowths of mucous membrane growing outward for the antrum, upward for the frontal, backward into the sphenoidal turbinate for the sphenoidal, and into the lateral mass for the ethmoidal sinuses. While it has been generally stated that the antrum shows in the fourth month of fetal life, he found in some dissections of fetuses of three to four months the antrum and ethmoidal sinuses in an advanced stage of development and the beginning of the outgrowths of the sphenoidal and frontal well shown at the same period. As the sinuses grow from the mucous membrane coming from the ethmoidal bone, the antrum should be considered as a large ethmoidal cell, the same as are the frontal and sphenoidal sinuses.

It has been generally stated that the antrum is supplied with filaments of nerves from the second division of the fifth nerve, through Meckel's ganglion, distributing branches to the inner wall of the antrum through bony canals. The author, however, thinks this is probably not true, but that the whole of the nerve supply enters through the ostia of the different sinuses, being carried in by the mucous membrane. He has tried to find that the sinuses are supplied by nerves other than or in addition to those that enter by the ostia of the sinuses, but has failed to find anything beyond the assertion that such is the case. He thinks that the mucous membrane carries its nerve supply with it wherever it spreads. In an examination of a large number of skul's he did not find any cell to contain two ostia, not even a large frontal which spread from side to side with only a partial partition between; when this was the case, there was only one drainage ostium into one side of the nose, which is proof that the cell grew from only one side. The position of the partition between adjacent cells depends upon the quickness of one cell to outgrow another, as seen in the varied partition of the septum dividing the two frontal sinuses. One nearly always outgrows the other, notwithstanding the fact that the frontal bones are closed by a suture in the center, which would seem to mark off the limit of each frontal sinus. One vigorously growing sinus, however, finds no difficulty in trespassing on the area of the other and to such an extent that we find only one frontal sinus opening into one side of the nose, yet so large that it completely crowds out the other, which in these cases remains an ethmoidal cell, and the septum which ought to be a frontal septum is in reality a frontal-ethmoidal septum.

The position of the ostia of these cells depends upon the place at which the mucous membrane begins to bud out, or pit, the favorite place being the upper part of the unciform groove, at the upper part of the groove of the bulla and the posterior part of the superior meatus. The pitting may begin at any part of the groove between the basal lamella, but these are the favorite places for the formation of the buds.

Most of the anterior ethmoidal cells open into the upper part of the groove of the unciform, the remainder into the groove of the ethmoid bulla. In the upper part of the groove of the bulla are the so-called accessory turbinals; there are three, and between each spring cells, usually three in all, one being the cell of the bulla, which is almost always constant.

In the fourteen babies' heads that were dissected, about the same number of cells were found as in the adult. There were no cells that one could say were certainly sphenoid cells, but in every case one could easily find the future frontal sinus growing up into position. Not only were the cells in number ranging from nine to fourteen found in the eighteen dissections, but the same number and arrangement were found in fetuses of three and a half months, four, five, six and seven months. As growth goes on, the gradual tendency is for the upper turbinates or folds to disappear by the merging of two or three into one. When this does not take place, we have the extra turbinate of the adult.

The method of dissection was to do it from the orbital side, the mucous membrane being carefully dissected from the cell walls and the bone broken off bit by bit so as to expose the whole of the antrum and the ethmoidal cells. The mucous membrane peeled off easily, and with care it was possible to have it stand out uninjured and in the same shape that it was when held by its bony or cartilaginous walls. In this way it could be easily seen into which meatus the cell opened, as each cell was held into the nasal mucous membrane by its stalk-opening, so that it stood out like a grape. *Richards.*

A New Method of External Frontal Sinus Operation Without Deformity.

Joseph C. Beck, Chicago (*Journal of A. M. A.*, August 8, 1908). Dr. Beck's method makes use of the osteoplastic flap in the following manner:

A radiogram is first made, so as to outline the sinuses, and from this radiogram a celluloid tracing is made and sterilized, so as to be used during the operation. An incision is made through the skin and subcutaneous tissues through the upper margins of the eyebrows, then downward and inward, as is usually done in the Killian operation. The saw is then carefully drawn from within outwards a few strokes, so as to sever the bone, but not the periosteum. Care must be taken not to saw through the entire thickness of the bone, as it will readily break when it is inverted downward over the nose. The skin flap is now reflected upward and the periosteal bone flap downward, exposing both sinuses. If only one sinus is to be exposed, the technic varies only in the osteoperiosteal flap, the incision being made within the limits of the frontal sinus septum. The skin flap may be made by making a perpendicular incision from the internal angle of the orbit as high as the radiogram indiates the height of the frontal sinus, within the natural intersuperciliary furrow, or wrinkle.

The diseased mucous membrane is then thoroughly eradicated, but not curetted, the natural opening into the nose enlarged, the anterior ethmoidal cells removed, a large rubber tube introduced, through which is passed a wick, the wicking being loosely folded within the cavity of the frontal sinus, and the other end of the tube passed down into the nose close to the floor, with a small portion of the wicking protruding.

The osteoplastic flap is then replaced, the skin flap brought down and sutured with silkworm gut, using the Halstead subdermal suture with a few horse hairs right over the bridge of the nose. The gauze is removed the next day, and on the third or fifth day the rubber tube is replaced by a silver filigree or gold tube. Douches or strong blowing of the nose should be avoided immediately after the operation. *Richards.*

Report Upon Three Interesting Cases of Sinusitis Associated with Serious Eye Complications.

Alfred Wiener, New York (*American Journal of Surgery*, October, 1908). These cases illustrate the recent publications of various authors, such as A. Knapp, Posey and Onodi, of the close association which diseases of the intranasal passages, especially that of the sinuses, play in connection with some of the more serious affections of the eye.

In Case 1, there was marked exophthalmus, and on operation an empyema of the frontal sinus and an extradural abscess were found.

In Case 2, absolute blindness followed an attack of rhinopharyngitis. The middle turbinate bone was resected and the post-ethmoidal cells were curetted first on the right side and then on the left, with evidence of disease at each operation. Vision returned in one eye, but not at all in the other.

The author regards the condition to have been an acute retrobulbar neuritis occurring in the canalicular portion of the optic nerve, brought about through a posterior ethmoiditis on both sides and probably of syphilitic origin.

Case 3 presented complete blindness in a child of nine, following an acute coryza and severe frontal headache. Vision returned in about two weeks after mild local treatment of the nose was instituted. *Richards.*

Histologic Changes in the Nasal Mucous Membrane in Cases of Leukemia, and the Cause of Nasal Hemorrhage in This Disease.

OTTO MAYER (*Monatsschrift f. Ohrenheilkunde,* etc., vol. xlii, Heft 5, p. 259) studied histologically the nasal condition of a woman who had died from pernicious leukemia. From a study of this case he says that the nasal bleeding in leukemic people is probably produced in somewhat the following manner: The leukemic infiltration of the mucous membrane produces an itching which causes the patient to dig into the nose, wounding the septal mucous membrane. Because the superficial blood vessels are dilated this injury gives rise to a free flow of blood. Further, the injury, on account of leukemic condition of the tissues, does not heal but develops into an ulcer with considerable loss of tissue. The richness of the blood vessels in the base of the ulcer accounts for the marked bleeding on the slightest trauma. *Wood.*

Extradural Abscess With Disturbance of Speech.

WILHELM JANSEN (*Monat. f. Ohrenheilk.*, etc., vol. xlii, Heft 6, 1908, p. 304) reports a case of a man 21 years old, who came to him September 14th with suppuration of the mastoid antrum. He was treated until September 25th, when operation was decided upon, but the next day, before the operation, he suddenly developed a disturbance of speech which made him apparently unable to understand his surroundings and the nurses, and to give false and incomprehensible answers to questions. The antrum was found full of pus, but no fistula leading to the middle cerebral fossa was discovered. The middle fossa, however, was opened, and from beneath the dura a tablespoonful of odorless pus was evacuated. The dura was normal. After division of the dura the brain was punctured with a knife in several directions but no pus found. The next day after the operation the patient's general condition was better, although aphasia was still present. The disturbance of speech gradually disappeared in about eight days. *Wood.*

Acute Suppuration of the Sphenoidal Sinus, With Intracranial and Orbital Complication.

G. Trautman (*Archiv. f. Lar. u. Rhin.*, Bd. 20, Heft 3) reviews the literature of the subject, and reports a case characterized by general septicemia, with empyema of the sphenoidal sinus, in association with purulent rhinitis and purulent phlebitis of the cavernous sinus, with beginning purulent hemorrhagic basal meningitis. A fatal termination occurred after five days' illness, and at the autopsy, in addition to the above conditions, numerous subpleural abscessses of the lungs were found, with rupture into the pleura. There was also swelling of the spleen with clouding and miliary abscess of the kidneys, beginning fatty degeneration of the liver and caseation of the bronchial glands. *Goodale.*

On the Relation of Syphilis to Ozena.

Frese (*Archiv. f. Lar. u. Rhin.*, Bd. 20. Heft 3) reviews the history of sixty-one cases of ozena with reference to etiology, and finds that in only 26.2 per cent was it possible to demonstrate definitely hereditary syphilis. In the remaining cases a positive determination of the etiology was not possible. Nevertheless, the author believes that a large proportion of ozena cases can be referred to a definite cause, and he regards syphilis as the most probable factor in the causation of the disease.

The explanation of this development may be in the first place a general lowering of the constitution (dyscrasia), or secondly ozena may be regarded as a metasyphilitic affection. Neither of these explanations seems adequate. Finally we might attribute ozena to a direct local injury of the mucous membrane through the syphilitic virus, and this last hypothesis appears to be the most probable one. Syphilitic coryza does not, however, lead in all cases to ozena, and perhaps the duration and intensity of the syphilitic nasal process is of importance in this relation. Ozena in hereditary syphilis finds its analog in the post-syphilitic ozena, such as is manifested after the tertiary processes in the nose. *Goodale.*

The Importance of the Roentgen Rays in Rhinology.

Scheier (*Deutsche med. Wochenschrift,* No. 41, 1908). X-Ray examinations of the accessory sinuses of the nose, particularly the frontal sinus are of great importance, because the presence or absence of the sinus can be determined in this way. It is important to find out in regard to the size of the frontal sinus, how far upward it extends, how far posteriorly and the thickness of the anterior wall.

All these points can often be determined with the fluoroscope alone. X-Ray examinations were also found useful in determining diseased conditions of the accessory sinuses. Repeated examinations showed that a shadow was caused mainly by a collection of pus.

A direct radioscopy should be made first. This shows the lateral position of not only the frontal sinus, with its depth and the thiskness of the anterior wall, but also the size of the maxillary and in the majority of the cases of the sphenoid sinus. In sharply defined pictures, the height of the sphenoid sinus can be determined, and often the semi-circular projection of the sella turcica.

If the patient is then turned around, and the sinuses on the both sides compared, collections of pus can frequently be discovered by the difference in the clearness of the sinuses.

In taking radiographs the lateral position of the head will give the vertical and horizontal dimensions of the sinuses, while an antero-posterior exposure will show the maxillary, frontal sinuses and ethnoid cells separately and sharply defined, as well as the lateral extension of the frontal sinuses, the position of the septum and the separate frontal and orbital recesses.

The sagittal views are taken with the patient in the recumbent position with the face on the photographic plate. The author prefers the sitting position and has had better results. The patient is seated in a strong fixed chair, and the plate, which is covered with black paper, is placed against the forehead and nose, and held in position by a few turns of a gauze bandage.

In order to obtain a correct idea of pathologic conditions of the sinuses, it is important to study normal radiographs of the head.

Pus in a sinus shows very clearly on the picture, and even thin fluid contents in the sinuses offer relatively great resistance to the X-Rays. Thick pus will of course give a darker shadow.

Spiess watching the position of the instrument through the fluoroscope, has advocated opening the frontal sinus through the nose with an electrically driven trephine. The dangers of this method are so obvious that it has not been generally adopted.

In conclusion, the author states that skiagraphy as a diagnostic aid has a distinct place in rhinology, and will clear up many conditions formerly obscure. *Theisen.*

A Maxillary Sinusitis and Osteitis.

Moure and Brindel (*Revue Hebdomadaire de Laryngologie, D'Otologie,* January 4th, 1908) believe that all cases of chronic antrum disease, which have resisted medical treatment for three to four weeks should be subject to the radical operation, and recommend the Caldwell-Luc method in the great majority of the cases. In 246 out of 270 operated on by them, the disease was limited to the mucous membrane. This was at times partly inflamed; more or less thickened. In other cases it was degenerated, edematous and with small prominences. At times they found it distinctly polypoid. Finally, in some cases they met with the membrane of the paradental cyst. For all these forms of disease they believe in a thorough curettage. In the remaining 24 cases, disease of the bony wall was present either a simple osteitis or a necrosis, with or without the presence of sequestra, a fungous osteitis, or finally in one case a hypertrophic osteitis. This disease of the bone can depend upon some general diathesis. In 3 of the 24 cases it was shown that syphilis was the cause. In four of these only was was this suspected before. The diagnosis in the other seven was made at the time of the operation or several days following. In cases of syphilitic origin, there is always wont to be an unusually profuse hemorrhage during the operation, after the removal of the mucosa. It was easy to recognize the inflamed bone. In five of the eleven cases more than simple osteitis was present. In two of these, sequestra were discovered. In one case the sequestra had been the cause of a persistent neuralgia which had defied all attempts at relief. In the three other cases a necrosis of the antrum wall was discovered, on the internal wall in the neighborhood of the infundibulum, and once on the postoral wall. One patient showed a pronounced thickening of all the walls of the sinus. Besides syphilis, the authors point out as other possible causes, virulence of infection of the mucous membrane (three cases); ethmoiditis and sphenoiditis (four cases); presence of mucous polyps in the cavity (one case). Five cases were due to irritation from a paradental cyst. The authors conclude as follows:

First, that the osteitis which accompanies at times chronic suppuration of the antrum is to be regarded as a benign complication. Second, that it is of greatest importance to recognize accurately the cause of the osteitis and to apply the proper treatment. Third, that this osteitis will permit the discovery, in certain cases, of a general unrecognized affection, ignorance of which would be of great disadvantage to the patient, especially if it is of a syphilitic nature.

Harris.

The Present State of the Radical Operation for Empyema of the Sphenoidal Sinus, With Demonstration of a New Instrument.

R. H. SKILLERN (*Archiv. f. Lar. u. Rhin.*, Bd. 20, Heft 3) reports the different methods of entering the sphenoidal sinus, which may be divided into the extranasal and the endonasal. Of the extranasal we have the frontal, maxillary and oral; of the endonasal the direct and the indirect. The writer prefers the endonasal direct method, with previous resection of the middle turbinate, and simultaneous removal of the posterior ethmoid cells, by which the whole field of operation is visible. The details of the operation should be read in the original.

Goodale.

Anosmia Caused by Abscess of the Temporal Lobe.

BLOCH AND JULIUS HECHINGER (*Archiv. f. Ohrenheilk.*, Bd. 76, Heft 1 and 2, p. 32) review two published cases of anosmia following abscess in the temporal lobe, and report one case of their own. In two of the cases the anosmia occurred on the same side as the abscess, but in one of the reviewed cases there was crossed anosmia. In only one case was the presence of the anosmia discovered before the operation: in the other two cases the lack of smell was only determined after the operation, but it seemed most positive that the lack of smell was a consequence of the abscess and not of the operation. *Wood.*

Primary Tumors of the Sinuses of the Nose.

CITELLI (*Archiv. International de Laryngologie,* January, 1908), in connection with his colleague has covered this important subject in a very thorough way. Morgagni was the first to discover such a growth. Since then many anatomists have studied the subject, including Zuckerkandl. It is common to confuse secondary with primary growths which greatly alters the statistics. The author considers the various sinuses in order. He divides the tumors into benign, intermediary and malignant. Of the benign tumors there are the fibroma, soft and hard, the osteofibroma, the lipoma and the angioma. The most common are the soft fibromata. These are usually called mucous polyps. They resemble the nasal polyp but are wont to be single and one-sided. Their origin is obscure, but some irritation or individual predisposition is most probably at the bottom. The other benign neoplasms are rare. Two cases of angiomata and one of lipoma are all that there are on record. In addition to these benign tumors, Citelli makes a class of intermediary tumors. Of these the osteomata are the most common. The cause of them is similar to that of the polyps. While inflammation is undoubtedly the cause in certain cases, it must be remembered that in spite of the great frequency of inflammation in the nose, osteoma is comparatively rare. Enchondroma is exceedingly rare in the antrum. The two most typical cases are those of Heyfelder and Denuce. In all these intermediary tumors it is necessary to distinguish a stage of latency and a stage of manifestation. During the former period the diagnosis is difficult and the symptoms are inclined to be vague. Malignant tumors include endothelioma, sarcoma and carcinoma. Endothelioma is rare but Citelli has collected thirteen cases in the literature, and thinks that others would be reported if a careful histologic examination were made in every case of antrum growth. Sarcoma is not so common as is generally supposed. The majority of them are secondary, having their origin in the adjoining tissues. All varieties have been observed. Carcinoma is the most frequently met with of all the malignant growths. They spring either from the epithelium or from the glands in the mucosa.

The frontal sinus is much less commonly affected by tumors. Fibroma is very unusual. Osteoma is more often met with. Citelli is of the opinion that fifty cases will represent the total number. They spring usually from the anterior wall, and occur in people from twenty to thirty. As causes, the most probable are a special congenital predisposition and inflammation, especially traumatism. Until the tumor makes itself in evidence by causing tumefaction it is difficult or impossible to make a diagnosis. Finally, malignant growths of the frontal sinus are exceedingly infrequent. Citelli has records of one case of endothelioma, one case of carcinoma and six or eight cases of sarcoma. The sphenoid is still more rarely affected. A few cases of osteoma, one of endothelioma, one of carcinoma, and half dozen cases of sarcoma represent what have been reported. *Harris.*

Foreign Bodies in the Nasal Cavity as a Cause of Maxillary Empyema.

Krebs, Hildesheim (*Archives of Otology,* Vol. xxxvii, Nos. 3 and 4). The author reports two cases, in one of which an infant's rubber nipple had been in a child's nose for at least seven years. In the other case, that of a man aged 33, whose antra were known to be clear, while working a threshing machine a grain of corn flew into the left nostril. One year later a granuloma, to which was attached a grain of corn, was removed from the man's nostril. After two weeks' irrigation the antral suppuration ceased. *Campbell.*

Neuroses of the Nose.

Charles P. Grayson, Philadelphia (*Medical Record,* December 12, 1908), does not consider that laboratory studies of the secretions of the hay fever sufferer have yielded any results that are genuinely practical. It is of infinitely greater importance to remember and to remove the basic nutritive fault that is accountable for such alterations. *Richards.*

The Submucous Septal Operation.

O. Freer (*Archiv. f. Lar. u. Rhin.*, Bd. 20, Heft 3) describes the application of his method to different types of septal deviation, and to the numerous abnormalities which may be encountered. The original paper should be consulted for the details. *Goodale.*

Bleeding Polyp of the Septum.

G. Trautman (*Archiv. f. Lar. u. Rhin.*, Bd. 20, Heft 3) Clinical and histological reports of 3 cases. *Goodale.*

III.—PHARYNX.

Some Points in the Applied Anatomy of the Tonsil.

Harry A. Barnes, Boston (*The Boston Medical and Surgical Journal,* Sept. 24, 1908). This is an anatomical study. The capsule of the tonsil may be considered as a highly developed tunica propria of the faucial mucous membrane, separated from its epithelium by the development of the lymphoid nodules which compose the mass of the tonsil. As might be expected from this derivation, it completely invests all parts of the tonsil not covered by epithelium and is continuous with the tunica propria of the faucial mucosa. In microscopic sections it appears as a compact layer of fibrous tissue with numerous elastic and striated muscle fibers interspersed. A number of trabeculae of the same composition, except that muscle fibers are only occasionally found, are given off from its internal surface and extend into the substance of the tonsil between the crypts, and separate it into lobes. Some of the muscle fibers are from the superior constrictor and from the pillars. Blood vessels appear at intervals, the larger of which are seen at the junction of the trabeculae with the main capsule, from which point they supply the tonsil.

The capsule is thin, but tough, not over ½ mm. in thickness, and is quite easily separated from the surrounding tissues at its upper half, but is much more adherent at its inferior half, and it is at this point that the larger vessels enter the tonsil. Its structure, its toughness, and the ease with which it may be separated from the underlying tissues, make the operation of dissection of the tonsil "in capsule" a very practical one.

Richards.

Results in Adenoid and Tonsil Operations.

J. Payson Clark, Bŏston (*Boston Medical and Surgical Journal,* Sept. 24, 1908). This is a study made four years after operation of a series of cases operated upon for tonsils and adenoids, which were done in a public hospital. Of 595 cases which he attempted to look up, only 73 could be gotten hold of. In 35 of these cases mouth breathing was said to persist in some proportion. In three cases the mouth breathing was evidently due to obstruction, caused by a deviated septum, and in five others to hypertrophied turbinates. In only one case was there sufficient adenoid present to call for a second operation. In the rest of the cases in which mouth breathing persisted no obstruction could be found to account for it. Twenty-one still continued to snore more or less. Four cases in which enuresis existed were not relieved of this symptom by the operation. Twenty-eight cases were said to still have colds. In 60 of the cases tonsillar tissue was still visible, although in the large majority of the cases the tonsils had been quite thoroughly removed at the original operation. Only ten of the cases had tonsillitis, and of these ten, eight had only one attack. Forty-nine cases had given a definite history of tonsillitis before the operation. In about half of the cases there were remains of adenoid tissue in the vault, but not enough to require reoperation. *Richards.*

Lymphosarcoma of the Tonsil.

J. L. Goodale, Boston (*Boston Medical and Surgical Journal,* Sept. 24, 1908). Patient was a woman, 35 years of age, with the left tonsil transformed into a firm, reddened mass about the size of a hickory nut, with deep extensive ulceration. The glands on that side below the angle of the jaw were enlarged, firm and tender. A portion of the growth was removed and examined by Dr. J. H. Wright, who reported lymphosarcoma of a rapidly proliferating type. Ether was given with the object of operating, but the growth had extended to the soft palate, infiltrating it, together with the base of the tongue, so that no operation was performed.

The author having observed in a previous case of Hodgkins' disease that improvement took place after staphylococcus injection, and recognizing the very close relationship between Hodgkins' disease and lymphosarcoma, he determined to utilize this idea in the treatment of the case. Turpentine was therefore injected into the legs with the object of making abscesses. Two minims were first injected without result, except that the sites of injection became rather red and painful. At the same time injections of staphylococcus aureus vaccine were given every three days. The turpentine was increased every two days up to twelve minims at each injection. In about one week the patient began to show improvement and the whole condition was better. Ten days later she was talking easily, while the growth in the throat steadily diminished, and in about one month from the beginning had nearly disappeared. The turpentine injections, which had been borne without great discomfort, became very painful, and she began to have localized necrosis of the skin from the injections. Seven weeks from the time of beginning treatment there was a large stellate cicatrix at the site of the tonsil, and absolutely no sign of new growth, with disappearance of the infiltration at the base of the tongue and soft palate. The lymph nodes on the outside of the neck had become so small that only one about the size of a hickory nut was palpable. Six months from the beginning she was apparently as well as she had even been. On theoretical grounds the author is inclined to attribute the result to the vaccine, since the boils which he expected to excite by the turpentine did not appear until after the growth had nearly disappeared. *Richards.*

Case of Tonsillolith.

James M. Anders (*Journal of the American Medical Association*, April 11, 1908). The case is reported by the author because it is unique in some particulars. The patient, a man aged 59 years, had had several attacks of acute articular rheumatism. After his first attack he had suffered from attacks of tonsillitis for many years annually.

For three or four months before seen by the author, he had some pain and irritation around the right tonsil and in a corresponding part of the neck of the same side. For several weeks the local disturbance became much aggravated. There was great pain in swallowing, but solids could be more easily taken than liquids. His local physician had prescribed flax-seed poultices, and two days after starting to use them the patient became aware of the presence of a hard foreign body which projected into the throat. This caused severe coughing, and with the aid of frantic efforts on the part of the patient, a large mass, of calcareous hardness, was dislodged. There was practically no bleeding after the mass was expelled. An examination made three months after the tonsillolith had been expelled, revealed on palpation with the finger tip, a distinct depression between the palatine arches.

The weight of the tonsillolith is 189 grains, and it measured one and seven-eighths inches in its greatest diameter.

In the vast majority of the cases of tonsillar concretions, they are of medium size and cause no symptoms, but when they attain, as in the author's case, enormous dimensions, they produce irritation and even ulceration of the surrounding tissues.

The author's specimen so far as he has been able to determine is the largest on record.

Jackson reported one weighing 147 grains. The concretions are made up mostly of calcium carbonate and phosphate. They are the result of calcareous degeneration of retained tonsillar secretions. *Theisen.*

Streptococcic Infections of the Pharyngeal Adenoid Tissue in Adults.

ALICE G. BRYANT (*Journal American Medical Association,* June 13, 1908) states that this subject should receive most careful consideration at the hands of the laryngologist.

It is doubtful whether these streptococcic infections of the pharyngeal adenoid tissue in adults are clearly recognized by the general practitioner.

The paper is based upon ten cases in the writer's practice. It is most important to directly examine the post-nasal and nasal regions to settle any debated question where septic conditions are present that cannot be attributed clearly to some one definite region of the body.

The following illustrative case is reported:

Miss A., aged 30 years, had had an attack of influenza three months before seen by the writer. For two weeks there had been a rise in temperature each evening, ranging from 100.5° F. to 101.5° F., with slight elevation in the morning. She had been examined for pulmonary involvement with negative results.

A cough developed and masses of yellowish white opaque material were freely expelled. On examining the throat, the soft palate, uvula and posterior pillars and pharyngeal walls showed marked swelling and redness. The tonsils were swollen and slightly inflamed. On post-nasal examination the adenoid tissue covered with a creamy secretion was seen. The tissue looked "rat-eaten" and only one-third of it remained, and that was easily removed with the forceps.

Bacteriologic examination showed streptococci in abundance. Constitutional symptoms promptly disappeared after this treatment.

Two other cases are reported.

In none of the writer's cases were the ears involved nor was there any enlargement of the cervical glands.

Theisen.

Pathological Anatomy and Etiology of the Black Hairy Tongue.

H. Haenish (*Archiv. f. Lar. u. Rhin.*, Bd. 20. Heft 3) reviews the literature, and reports a case of this affection, exhibiting the characteristic hairy deposit.

The patient, a man 66 years of age, had noticed a peculiar sensation for three months on the palate, and fourteen days later discovered a black deposit on the tongue, in association with an acid taste. The hairy growths were removed, and replaced by a circumscribed yellow deposit. Examination of the hairs showed no growths of black fungi when cultivated, but numerous colonies of various cocci. Histologically the hairs were seen to be the hypertrophic and horny papillae filiform. The case is peculiar in showing no recurrence.

Goodale.

Torticollis as Complication of Adenotomy.

L. Neufeld (*Archiv. f. Lar. u. Rhin.*, Bd. 20, Heft 3) has observed this condition three times in the last five years. The torticollis appeared in one case two days and in another three days, and in a third five days after the operation. In all instances, the lymph nodes in the neck were enlarged and sensitive. Elevations of temperature were not noted after the first week. The general health of the children was fully established at the end of this time. Passive movements of the head, which at first were very painful, diminished at the end of a week. The head in the first few days was turned backward and sideways, later only sideways. The duration of this manifestation varied from fourteen days to two months. Complete recovery took place in all cases.

Goodale.

The Pigmentations of the Mucous Membrane of the Mouth.

G. H. ANTHONY (*Journal of the American Medical Association,* November 14, 1908) states that the pigmentations of the mucous membrane of the mouth have not been sufficiently studied.

Congenital pigmentation.—The most common form is the soft mole which may be simply a pigmentation of the skin or may present itself as a mammillated nodule with or without pigmentation or hairy growth.

Oppenheim has reported a case of soft mole affecting the conjunctiva of the eye ball and extending even over the cornea, but the writer could find no cases on record in which soft moles were present in the mucous membrane of the mouth. Even in cases in which the cutaneous surface was studded with pigmentary moles, the mucous membrane of the mouth was free. The author has observed one case of such growth occurring in the mucous membrane of the mouth.

In that case the nodule was situated in the middle line of the roof of the mouth on the hard palate in a woman aged 60 years. There were a few pigmentary moles of the skin also present.

Roy has reported a case of melanosis of the palate which after existing twenty years became sarcomatous.

The pigmentations of xeroderma pigmentosum affect the vermillion border of the lips, but they have not been observed in the mouth. The fibroma molluscum tumors of von Recklinghausen's disease frequently occur in the mouth, but the pigmentations affecting the mucous membrane must be quite rare, because the only mention of them in recent literature is the report of Oddo, who describes them in two cases as well-defined areas of pigmentation affecting various parts of the mucous membrane and resembling the pigmentations seen in the mouth of dogs.

The pigmentations of the mucous membrane in the mouth in Addison's disease are usually not the same shade of bronze-color as in the skin, but consist of circumscribed areas of melanotic pigmentation. They are rarely absent, and are of great diagnostic importance.

Stelwagon emphasizes the fact that pigmentation is frequently a lesion relic in scleroderma. Audry has reported a case of scleremia, in which ill-defined plaques of pigmentations were present in the mucous membrane of the cheeks.

Phthiriasis.—Thibieige and a number of others have observed melanotic pigmentations of the mucous membrane of the mouth in cachectic subjects with cutaneous brown pigmentation. The fact that such buccal pigmentations occur only in cachectic subjects suggests the thought that hemochromatosis is the cause of such pigmentations rather than pediculi per se.

Osler states that whenever hemochromatosis, either with or without diabetes, is suspected, the correctness of the diagnosis intra vitam will be made much more probable by removal of portions of the pigmented skin and the finding (by the potassium ferrocyanid test) of iron-containing pigment in the cells of the sweat glands, and of the ochre-yellow in the muscle fibres of any blood vessels that may be present, by the use of hemofuchsin.

That pigmentation is actually caused by pediculi is shown by the fact that pediculi pubis cause blue pigmentations of the skin, so that with hemochromatosis excluded, it is extremely probable that pediculi corporis produce brown pigmentations of the skin, and black pigmentation of the mucous membrane of the mouth. In the class of anomalous pigmentations, the author includes all pigmentations of the mucous membrane of the mouth which can not be assigned to any of the preceding disorders.

These pigmentations are almost invariably located in the mucous membrane of the lips. They are usually observed in patients suffering from gastrointestinal autointoxication, diabetes, and neurasthenia. *Theisen.*

IV.—LARYNX.

Clinical Contributions to the Significance of Esophagoscopy and Tracheoscopy in Their Diagnostic and Therapeutic Relation.

E. Schmiegelow (*Archiv. f. Lar. u. Rhin.* Bd. 20, Heft 3) reports a series of cases where the direct inspection of the esophagus, the trachea and the bronchial tubes, was of great value in clinical and therapeutic relation. Cases of cancer of the esophagus, spastic stricture and dilatations came under observation. The writer distinguishes between diffuse and circumscribed dilatations or diverticula. The diffuse dilatations are, as a rule, to be attributed to cicatricial cancerous stenosis, which prevents the food from passing down, and causes it to be heaped up above, with the result of dilating the esophagus. These retention dilatations seldom reach a large size, and may even be absent in spite of the stenosis, as long as the muscular apparatus of the tube above is sufficiently powerful. The dilatation is greatest immediately above the stenosis, and diminishes towards the pharynx.

In addition, there may rarely be found enormous dilatations of the esophagus, without stenosis. These usually assume a spindle form, and may attain the diameter of a man's arm. These dilatations are to be considered as a neurosis with paresis of the esophageal muscles, perhaps in association with spasm of the muscles of the cordia.

The writer reports cases of Zenker's pulsion-diverticulum and of the Rokitansky's traction-diverticulum.

Goodale.

Amputation of Epiglottis in Persons Suffering From Tuberculosis of the Larynx.

Moeller (*Revue Hebdomadaire de Laryngologie, D'Otologie,* April 18th, 1908). While removal of the epiglottis has occasionally been done in whole or part for extensive disease, Hajek was the first, in 1893, to do a complete operation on a consumptive suffering from dyspnea. Moritz Schmidt has performed it in nine cases, in three of whom there was a perfect cure. The author has performed the operation ten times, and recommends as the best instrument to perform it, the guillotine devised by Alexander, with which he claims it can be easily performed. He has never seen any severe consequences from the operation. If one does not touch anything but the middle of the epiglottis, there is no considerable bleeding. There is some sore throat for a few days. In three of the ten times thus operated upon, a cure was effected. Two of the cases died later, one of cerebral tuberculosis. He did not have any dysphagia after the operation. The other died from the pulmonary disease. Two cases are still under treatment, and in four an accurate report can not be made. In three cases suffering from dysphagia there was entire relief. In the author's opinion, the indications for the operation are first, a tubercular lesion, situated in the free part of the epiglottis, alone, provided the patient's condition is such as to permit of this slight intervention; second, a pronounced dysphagia in a case of laryngeal tuberculosis without regard to the condition of the rest of the larynx and the lungs, provided that the dysphagia is caused by a lesion of the epiglottis; third, a severe tubercular affection of the epiglottis, even without dysphagia in a case of laryngeal tuberculosis, provided that there is no pulmonary disease or the condition of the lungs is such that one can hope for a complete recovery or a considerable improvement. In these cases the removal of the epiglottis makes the treatment of the disease in the interior of the larynx easier, so that one may hope for a complete cure. *Harris.*

The Phonation Center in the Cortex of the Dog.

J. KATZENSTEIN (*Archiv. f. Lar. u. Rhin.*, Bd. 20, Heft 3). The origin of voice production in the dog may be conceived as follows: The anterior central gyrus, the precentral gyrus, the anterior gyrus compositus, i. e., the parts of the cerebrum from which proceed the movements of the abdominal muscles, the diaphragm, the thorax, the larynx, the pharynx, the tongue, the lips and the jaw form the lower or primary center for the component muscles of voice production. At a definite point of the above-mentioned cortical field, namely the anterior central gyrus, which is at the same time the center of cortical respiration, lies the center of a higher order for voice production, from which the whole act of voice is voluntarily produced. We find therefore, the primary lower centers of voice production, i. e., centers of perception for the movements of the abnormal muscles, the diaphragm, the thorax, larynx, pharynx, palate, tongue and lips, and the jaw in the above-mentioned centers of the anterior central gyrus, the precentral gyrus and the anterior gyrus compositus; on the other hand, the center for the production of the whole act of voice production, in which the above-mentioned portions of the vocal act unite in common with the respiratory center at a place in the total cortical field for voice production, is found in the anterior central gyrus. The production of voice proceeds therefore from the observations which the animal makes in the first period of his life during the act of expiration, and the simultaneous expiratory movement of the vocal cords. In the first weeks of life young dogs do not bark, they whine, whimper and cry, according to the sensations of pain or pleasure which they feel. After about six weeks, probably involuntarily, an expiratory act gives rise to a bark, and the animal seizes upon this act as a means of expressing later his sensations of pleasure and pain. *Goodale.*

A Report of Three Cases of Laryngeal Growth Removed by Direct Laryngoscopy.

JOHN McCOY, New York (*American Journal of Surgery*, September, 1908). The first case was a fibroma of the inner surface of the left cord, failure having resulted on every attempt to remove the growth by the indirect method. A hypodermic injection of 1/4 grain of morphia and chloroform anesthesia were administered. The growth was readily seen, but there was such excessive bronchorrhea and difficulty from the narcosis that a hypodermic injection of atropia gr. 1/100 had to be given. The separable speculum of Dr. Jackson was used. The growth was completely removed and the patient's voice quite restored.

In Case 2 there was a growth in the interarytenoid region, which proved to be hypertrophic tuberculosis. Morphia, atropia, cocain, chloroform anesthesia was used. The growth was pale in color and papillary in appearance, extending from just below the cords to the upper limit of the larynx. It was removed piece by piece with the straight forceps. The difficulties encountered in this case were, that the patient seemed to come out from deep anesthesia and struggle, which necessitated some delay in getting him well under again. Also, he did not seem to bear the combination of chloroform and cocain very well, as the pulse was more rapid and weak than it should have been.

Case 3 was a chronic inflammatory thickening at the base of the left arytenoid, and was removed without particular difficulty.

The author thinks that the combination of morphin and atropin seems to control any tendency to excessive bronchial secretion. *Richards.*

Laryngeal Crises and Paresis of the Abductors of the Vocal Cords as Important Early Symptoms of Tabes.

Otto T. Freer, Chicago (*Journal A. M. A.*, Sept. 5, 1908). "The laryngeal paralyses found in tabes, though often the first and possibly the only paralytic symptoms to occur, are but a portion of a group of paralyses found as results of pathologic degenerative changes in the cranial nerves or their medullary origin. Thus paralyses of the ocular muscles, the levator palati, the trapezius or sternomastoid muscles may be found. Indeed, the paralyses due to cranial nerve involvement in tabes may be so numerous and extensive that bulbar paralysis is simulated.

"The degenerative changes found in the laryngeal paralyses of tabes may be found in the nucleus of the vago-accessorius nerve, or in the intrabulbar root fibers of the vagus and accessorius, or in their peripheral extensions. The degeneration may also be limited to the vagus trunk or that of the recurrens, the medullary portions of the nerves being intact."

A case is reported in detail, illustrating the laryngeal paralyses of tabes. *Richards.*

Primary Lupus of the Larynx.

A. Rosenberg (*Arch. f. Lar. u. Rhin.*, Bd. 20, Heft 3) reports two cases of this affection, with special reference to differential diagnosis. Papilloma is readily excluded, being a rose-colored, cauliflower-like, often pedunculated growth, movable with the probe, and almost never being situated on the epiglottis. Syphilis in the stage of infiltration or ulceration is readily separable, exhibiting more congestion, and appearing moister, more shining and at times edematous, while the development is more rapid, and leads to marked thickening of the epiglottis. Syphilitic ulcerations are more sharply bordered and deeper, the surrounding tissue is more congested, brighter red and moister. If the lupus process has gone to cicatrization, it may be confused with syphilitic scars, although these latter usually show more a fibrous, dense and white appearance, and also extend into the depths. A special characteristic of lupus is the presence of the three stages of infiltration ulceration and cicatrization, especially the formation of brownish-red nodules in the scars.

In distinction from tuberculosis, it is first to be noted that lupus always attacks the epiglottis, and exhibits a granular or irregular surface of a moderately red color, with an increase in thickness greatly exceeding that present in tuberculosis. The aryepiglotic folds in lupus exhibit more symmetrical thickening, not translucent, but denser and slightly irregular, while finally lupus excites no pain or discomfort.

Goodale.

Pharyngeal and Laryngeal Syphilis.

T. Heryng (*Wiener medicinische Wochenschrift,* Nos. 44 and 45, 1908). Syphilitic angina may be recognized by a diffuse (more rarely localized), dark red or bluish-red color of the mucous membrane of the pharynx. The typical patches are seen most frequently on the tonsils, the uvula and the anterior faucial pillars. They at first have the appearance of a thin white deposit which can be better seen with natural light. As the process develops, irregular losses of tissue occur on the pillars and tonsils. Although these patches have a characteristic appearance, they are sometimes treated for chronic diphtheria and follicular tonsillitis.

A beginning pharyngeal lues is also commonly diagnosed as herpes, stomatitis, and pharyngo-mycosis.

A localization that is not always recognized is the lateral luetic pharyngitis. In the cases observed by the author lateral yellowish-red elevated patches, with swelling of the posterior pillars of the fauces were present. Small nodules develop on these elevated areas, and when thy break down, small ulcers, funnel shaped depressions, from 2-3 mm. deep, develop. In from 8 to 15 days these ulcers heal, leaving a smooth cicaricial surface. The pharyngeal syphiloma occurs either on the uvula, at times on its posterior surface, but more frequently on the posterior pharyngeal wall. It is either solitary or multiple. At first this takes the form of an either diffuse or nodular waxy infiltration which soon takes on a yellowish color and results in an ulcer with sharply defined edges.

This ulcerative process often extends to the tonsils and pillars, cicatrizing in one place and breaking out in another and may last for months.

The gummi of the velum results in perforations which sometimes develop very rapidly. A fistula communicating with the naso-pharynx develops in the middle of the infiltrated area. These perforating ulcers occur also on the hard palate, and result in necrosis of the bone. Ulcers involving the uvula often result in adhesions between the soft palate and posterior pharyngeal. wall Ulcerative processes involving the pharynx at times develop very insidiously without producing much pain and difficulty in swallowing.

In considering laryngeal syphilis the author states that the symptoms in the majority of the cases come on with disease of other organs, i. e., skin lesions, and involvement of the bones or glands.

The typical papule, the broad, gray condyloma, develops most frequently on the edge of the epiglottis, then the cords, the posterior wall of the larynx and the aryepiglottic ligaments in the order named.

The location and development of the tertiary infiltrations varies very much. They usually occur in from three to eight years after the primary lesion. Syphilitic corditis is usually unilateral, while corditis inferior is more frequently bilateral. Syphilitic laryngeal tumors, which at times are very large, may simulate malignant growths. The symptoms of secondary syphilis of the larynx are usually hoarseness, with a good deal of irritation and cough. Dangerous symptoms are caused by the breaking down of the specific infiltration and the formation of cicatrices and stenoses.

Considering the differential diagnosis the author states that the greatest difficulty is presented by the combination of syphilis and tuberculosis which frequently simulate carcinoma.

Syphilitic ulcers have certain characteristics, the sharply defined edges, the grayish base, the greatly reddened infiltrated surrounding tissue, which distinguish them from other ulcerative processes. The surest diagnostic evidence is however the rapid improvement when proper treatment is started.

Gummata rarely occur in the muscles of the larynx, although cases have been reported by Eisenberg, Turck and Mackenzie.

Syphilitic perichondritis attacks the cricoid cartilage most frequently, more rarely the arytenoid.

The edema that sometimes accompanies perichondritis may cause alarming symptoms, and patients with this condition should be carefully watched.

Treatment: The author recommends inunctions and intramuscular injections. In using inunctions he states that the nearer the seat of the lesion the applications can be made the more effective they will be. Intra-muscular injections are particularly recomended when a rapid course of the disease is feared, or when dangerous complications such as brain syphilis and destructions of important organs occur.

Schwinner's sozoidol of mercury is the best for intramuscular injections. The formula is Hydr. sozoidolic, 2.0-4.0 Kal. iod. 3.0-6.0, Aq. destill. steril. 5-0.

In considering the use of iodide of potash he mentions the fact that laryngeal edema may be produced, and for this reason it should be used with the greatest care in patients with kidney or heart trouble. *Theisen.*

Needle in the Left Bronchus.

SCHROTTER (*Deutsche medicinische Wochenschrift,* April 9, 1908) has operated upon 10 cases of foreign bodies in the trachea and bronchi, and reports another case of an unusually long needle in the bronchus of a 12-year-old child.

While the child was holding a shawl pin in the mouth a sudden paroxysm of coughing followed by a deep inspiration carried it into the air passages. This was followed by a severe paroxysm of coughing and cyanosis. The coughing continued during the day, but the child did not complain of any pain in the chest and slept nearly all night. Between the paroxysms of coughing which came on during the next few days, the child felt perfectly well. The child was sent to the hospital and an X-Ray examination made. The radiograph showed the presence of the pin in the left main bronchus, the head of the pin being down.

The operation was performed without general anesthesia, with the child in the sitting position. A tube with a diameter of 8.5 mm. and 22 cm. long, was easily passed into the trachea under the guidance of the finger.

The point of the pin could be seen projecting from the left bronchus into the trachea.

After two attempts the pin was successfully removed on the twenty-first day after it was aspirated. The operation was not followed by any reaction at all, and the child was able to leave the hospital on the third day. *Theisen.*

Observations on Diffuse Pachydermia Laryngis.

W. Albrecht (*Archiv. f. Lar. u. Rhin.*, Bd. 20, Heft 3) reports a case characterized by the formation of white, chalky masses, situated upon the vocal cords, consisting of a horny epithelium, in association with inflammatory reddening of the vocal cords. The author compares the manifestations present in his case with those reported by other observers. In spite of their clinical differences, all belong etiologically and pathologically to the same category. *Goodale.*

Tracheobronchoscopy for the Removal of Foreign Bodies.

C. Jackson (*Archiv. f. Lar. u. Rhin.* Bd. 20, Heft 3) describes his well-known apparatus, and its application in several cases. A number of observations of interest are made, among others the necessity of wearing glasses to avoid the danger of secretions from the bronchial tubes striking the eye. Even if the secretions are not likely to produce infection of the operator, yet it is important to prevent the possibility that cocain may be coughed up and interfere with the vision. *Goodale.*

BOOK REVIEWS.

Bier's Hyperemic Treatment in Surgery, Medicine and all the Specialties: A Manual of Its Practical Application.

By Willy Meyer, M. D., Professor of Surgery at the New York Post-Graduate Medical School and Hospital; and Professor Dr. Victor Schmieden, Assistant to Professor Bier at Berlin University, Germany. Octavo of 209 pages, illustrated. Philadelphia and London: W. B. Saunders Company, 1908. Cloth, $3.00 net.

This volume is not a translation of Prof. Bier's work on hyperemia, but is rather a brochure detailing the principles, methods of inducing, and results of using hyperemic treatment, and was written in order to furnish those that cannot read German an authoritative English version of the work of Prof. Bier. The section devoted to rhinology, pharyngology, laryngology, discusses acute coryza, frontal and maxillary sinusitis, tonsillitis, catarrh of the upper air passages, hay-fever, chronic angina and tuberculosis of the larynx. The suction apparatuses for the nose and for the tonsils are illustrated by clear cuts. The book is well gotten up, the illustrations being especially commendable.

The Surgery of the Ear.

By Samuel J. Kopetzky, M. D., Attending Otologist, New York City Children's Hospital and Schools; Attending Otologist to the New York Red Cross Hospital; Assistant Surgeon and Instructor in Operative Surgery of the Ear, Manhattan Eye, Ear and Throat Hospital; Pathologist and Surgeon, New York Throat, Nose and Lung Hospital; Fellow of the American Laryngological, Rhinological and Otological Society; Fellow of the New York Academy of Medicine, American Medical Association, Otological Society of Germany, etc. Illustrated with 63 Half-Tone and Line Drawings, 8 Charts and 4 Colored Plates. Rebman Co., New York. Price, $4.00.

Although at present there is no lack of good text books on otology either in the English or most foreign languages, the work of Kopetzky's will be a welcome addition to the library of every specialist as well as many general practitioners who seek information regarding the operative procedures connected with the ear. That the reviewer does not always coincide with the opinions expressed by the author is natural in a branch so comparatively young as ear surgery. Yet every reader of the book will acknowledge the zeal and the large experience of the author. Space does not permit the review of every chapter, much as it might be desired.

The author in describing the "simple mastoid operation," is not in favor of the so-called "blood clot" method, as his results were not good. Most of us will agree with him that this question is still sub judice.

The account of mastoid operations on the infant and young child is very valuable, as Kopetzky's special experience in this particular is evident. The difference in the sections through the ears of the child and adult on plate 7 would have been still more striking, if cuts from the same side would have been made. This chapter, however, is one of the best in the book.

The thorough description of the anatomy of the different parts is often a tiresome task for the reader, but we are glad to state that Kopetzky has succeeded in making those parts easily readable. "A thorough knowledge of the anatomy of the parts and their inter-relations is conducive to thoroughness" is an expression carried out with fidelity. The references to the figures all through the book would have been of greater service if the number of the page on which it is found would have been stated. Some of the drawings are very good, but more colored plates would have been desirable. The book may be read advantageously by every practitioner interested in the subject. *W. F.*

Diseases of the Nose, Throat and Ear, Medical and Surgical.

By William Lincoln Ballenger, M. D., Professor of Otology, Rhinology and Laryngology, College of Physicians and Surgeons, Department of Medicine, University of Illinois; Fellow of the American Laryngological Association; Fellow of the American Laryngological, Rhinological and Otological Society, etc. Illustrated with 471 engravings and 16 plates. Lea and Febiger, Philadelphia and New York, 1908.

Modernity is the key note of Ballenger's treatise, if indeed it is not futurity. For he has evidently striven to give the newest developments, even if they have not been subjected to the searchlight of experience. Differing from most textbooks which are old before they are issued, this one at least has the merit of being new. While the author may be required to change some of his claims in future editions, this is no more than is done by those who write the more or less dull, more or less prosy, more or less conservative text-books which are so common nowadays.

There is also a sense of satisfaction which is experienced by one who studies the illustrations which are in the main original and which show what they are intended to bring out.

The descriptions of operations, especially those most recently devised, are worthy of commendation. Ballenger has the power of making clear even the most technical details of operative procedure ordinarily so difficult of understanding.

The letterpress is good, the type clear, the paper of good color and quality, in fact, taking the book as a whole it is most satisfactory. Some few typographical and grammatical oversights will doubtless be corrected in subsequent editions.

INDEX OF AUTHORS.

ALBRECHT, W., 1071.
Alderton, 1029.
Alexander, G., 498, 1030.
Allport, F., 250, 333.
Alt, F., 497.
Amberg, E., 1033.
Anders, J. M., 1059.
Anthony, G. H., 1062.

BALLIN, 249.
Barnes, H. A., 1057.
Barnhill, J. F., 736.
Beck, Jos. C., 265, 1048.
Benjamin, C. E., 1039.
Berliner, 851.
Bishop, L. F., 1037.
Blegvad, N. Rh., 753.
Bloch, 1054.
Braislin, 1030.
Brindel, 1053.
Brunk, 854.
Bryant, A. C., 1060.
Bryant, W. S., 163, 652, 847, 1035, 1044.
Burger, H., 502.

CABOCHE, HENRI, 180.
Canfield, R. B., 451.
Casselberry, W. E., 258, 607.
Citelli, 1055.
Clark, J. P., 787, 1058.
Cocks, 1027.
Coffin, L. A., 258.
Cohn, J., 502.
Conlon, 258.
Cott, G. F., 1044.
Crawford, J. H., 1036.
Curran, E. J., 1046.
Curtis, H. H., 854.

DABNEY, W. R., 439, 844.
Delavan, D. B., 631.
Dench, E. B., 746, 1028.
Denker, A., 959.

v. EICKEN, 853.

FERRAN, 1038.
Fiedler, M., 1038.
Fowler, 1030.
Freer, O., 1057, 1067.
Frese, 1051.
Freytag, 246.
Fridenberg, Percy, 140, 670.

GLEITSMANN, J. W., 587.
Goodale, J. L., 851, 1058.
Gould, G. M., 1036.
Grayson, C. P., 1056.
Grosvenor, L. N., 470.

HAENISH, H., 1061.
Hammond, P., 1044.
Harris, T. J., 645.
Haskin, W. H., 985.
Hartmann, 853.
Heath, 250.
Hechinger, J., 1054.
Hecht, 849.
Heimann, Theodor, 800.
Henkes, I. C., 505.
Heryng, Th., 1000, 1068.
Heydenrich, 852.
Hopkins, F. E., 1022.
Horn, 853.
Hubbard, T., 172.
Hudson-Makuen, G., 263.
Hunt, 247.

INGERSOLL, J. M., 901.
Isemer, F., 499.

JACKSON, CHEVALIER, 261, 263, 1071.
Jansen, A., 367.
Jansen, W., 1050.

KANASUGI, H. E., 1044.
Katzenstein, J., 1065.
Kerley, 257.
Kerrison, 847.
Killian, G., 502.
King, G., 178.
Kittredge, F. E., 495.
Knapp, 246.
Knapp, A., 1045.
Knause, 843.
Koblanck, 849.
Koellreutter, 245.
Kopetzky, S. J., 117.
Krebs, 1056.
Kuttner, 850.

LANGE, W., 496.
Levinger, 851.
Loeb, C., 1000.

McCOY, J., 1066.
McKernon, 1027.
Manasse, 244.
Massei, F., 931.
Mayer, O., 1050.
Mermod, 995.
Miller, Albert, 180.
Moeller, 1064.
Moll, 506.
Myers, H. L., 167.
Moure, 1053.

NEUFELD, 259, 1061.
Neurnberg, F., 1041.

ONODI, A., 1.
Oppenheimer, 1026.

PAGE, J. R., 1034.
Passow, A., 497.
Phillips, Wendell, 1031.

REARDON, 259.
Rethi, 849.
Robinson, Beverly, 850.
Rosenberg, A., 1067.
Roy, D., 1033.
Ruppert, 840.
Rutten, E., 1040, 1042.

SAUER, W. E., 431.
Scheier, 1052.
Scheven, 850.
Schmidt, 849.
Schmiegelow, E., 1064.
Schroetter, 853, 1070.
Scott, 846.
Sendziak, 852.
Shambaugh, J. E., 250, 251, 661.
Sheedy, 260.
Siebenmann, 245.
Skillern, R. H., 1054.
Smith, S. MacCuen, 723.
Stillman, E. R., 1036.
Stimson, 256.
Strass, H., 1.
Struycken, H. J. L., 499.
Stucky, J. A., 427.
Swain, H. L., 974.

THOMPSON, 247.
Trautman, G., 1051, 1057.

VOSS, 244, 1029.

WACHENHEIM, 260.
Wales, E. DeW., 398.
Wiener, A., 1033, 1049.
Winckler, E., 498.
Wittmaack, 243.
Wood., Geo. B., 261, 855.
Wright, 257.

YEARSLEY, 249.

ZEBROWSKI, 847.
Zimmerman, 848.

INDEX OF TITLES.

ABDUCTOR paralysis larynx, Casselberry. 607.
Abductor paralysis larynx, anatomy and physiology. Gleitsmann. 587.
Abductor paralysis larynx, in tabes Freer. 1067.
Abscess, brain—
  Amberg. 1033.
  Bloch and Hechinger. 1054.
  Dabney. 844.
  Heimann. 800.
  Kittredge. 495.
  Page. 1034.
  Stucky. 427.
  Thompson. 247.
Abscess. brain. associated with chronic purulent otitis media. Page. 1034.
Abscess, brain, diagnosis. Heimann. 800.
Abscess, brain. operation, Stucky. 427.
Abscess. brain. two fatal cases. Thompson. 247.
Abscess. extradural. with disturbance speech. Jansen. 1050.
Abscess. peritonsillar. Moll. 506.
Abscess. temporal lobe causing anosmia. Bloch and Hechinger. 1054.
Abscess. temporo-sphenoidal lobe complicating sinus thrombosis. Dabney. 844.
Abscesses. tongue. deep lying. Brunk. 854.
Abstracts from Current Otologic. Rhinologic and Laryngologic Literature. 243. 495. 841. 1026.
Accessory nasal cavities and optic nerve. Onodi. 1.
Accessory nasal disease. intracranial complications. Coffin. 258.
Accessory nasal cavities. surgical treatment orbital complications. Knapp. 1045.
Adenoid operations. complicated by torticollis. Neufeld. 1061.
Adenoid operations. results. Clark. 1058.
Adenoid patients. post-operative treatment Sheedy. 260.
Adenoids. late secondary hemorrhage following removal. Wachenheim. 260.
Adenoid tissue. adult. streptococcic infection. Bryant. 1060.
Adrenalin. influence in causation arteriosclerosis. Hopkins. 1022.
Air. hot. in otology. Ferran. 1038.
Air passages. upper. connection with genito-urinary system. Sendziak. 852.
Air passages. upper. treatment tuberculosis. Heryng. 1000.
Alveolar process. disease. Haskin. 985.
Analgesia nasal. prognostic symptom in dry catarrhal deafness. Roy. 1033.
Anatomy tonsils. Barnes. 1057.
Anesthesia. local. in operations on maxillary sinus. King. 178.
Anesthesia. local. intratympanic operations. Ballin. 249.
Aneurism internal carotid. fatal hemorrhage. Benjamin. 1039.
Angina. therapy. Berliner. 851
Anosmia. caused by abscess temporal lobe. Bloch and Hechinger. 1054.
Antrum operations. closure bone defects after Passow. 497.
Arteriosclerosis influence of adrenalin. Hopkins. 1022.

BACTERIOLOGY of suppurations temporal bone. Wales. 398.
Basilar membrane. piano-string theory. Bryant. 847.
Bier's hyperemia in acute middle ear suppuration. Isemer. 499.
Bone defect closure after antrum operations. Passow. 497.
Book reviews. 1072-1074
Brain abscess. associated with chronic purulent otitis media. Page. 1034.
Brain complications. mastoiditis. Knause. 843
Bronchoscopy. Jackson. 1071.
Bronchoscopy and foreign bodies. v. Eicken. 853.
Bronchus. foreign body. v. Schroetter. 853. 1070.

CARDIOVASCULAR disease. ear symptoms. Bishop. 1037.
Carotid. external. ligation in rhinology and pharyngology. Jackson. 261.
Carotid. internal. aneurism. Benjamin. 1039.
Cerebral abscess. Amberg. 1033.
Cholesteatoma. followed by sinus thrombosis. Knapp. 246.
Cholesteatoma middle ear and posterior cranial fossa. Zimmerman. 848.
Chondroma in faucial tonsils. Grosvenor. 470.
Cochlea. minute anatomy. Shambaugh. 251.
Cochlea. sensitiveness to tone impressions. Bryant. 1044.
Cranial fossa. middle. cholesteatoma. Zimmerman 848
Cryptogenetic infections. relation to pleuritis. Wood 855.
Cyst in inferior turbinate, purulent bone. Cohn. 502

DEAFNESS. chronic progressive labyrinthine. Manasse. 244.
Deafness. congenital. anatomy. Alt. 497.
Deafness. dry catarrhal and nasal analgesia. Roy. 1033

Deafness in course of acute osteomyelitis and septic processes in general, Siebenmann, 245.
Deafness, new-born, Koellreutter, 245.
Diphtheria, primary nasal—
  Hubbard, 172.
  Stimson, 256.
Dysmenorrhea, nasal, Kuttner, 850.

EAR deafness of new-born, disturbance in sound-conducting apparatus, Koellreutter, 1030.
Ear instrument, Fowler, 1030.
Ear lobule, recurrent keloid, Cocks, 1027.
Ear symptoms in cardiovascular disease, Bishop, 1037.
Ear treatment, conservative, Myers, 167.
Ear tuberculosis, Bryant, 1035.
Ear work, lactic acid bacilli cultures, Curtis, 854.
Encephalitis complicating mastoiditis—
  Bryant, 163.
  McKernon, 1027.
Epiglottis, amputation in tuberculosis of larynx, Moeller, 1064.
Epiglottis, cysts abscesses and edema Swain, 974.
Equilibrium disturbances in nonsuppurative diseases internal ear, Wittmaack, 243.
Equilibrium, static and dynamic, present knowledge organs, Fridenberg, 670.
Esophagoscopy, diagnostic and therapeutic relation, Schmiegelow, 1064.
Esophagoscopy, lamp for, Horn, 853.
Ethmoid cells at birth and development during fetal life, Curran, 1046.
Eustachian catarrh with hyperinflation Braislin, 1030.
Extradural abscess with disturbance of speech, Jansen 1050.
Eye complications of sinusitis, Wiener, 1049.

FACIAL nerve surgery, Beck, 265.
Facio-hypoglossal anastomosis, Cott, 1044.
Foreign body (needle) in left bronchus, Schrotter, 1070.
Foreign bodies nose cause of maxillary empyema, Krebs, 1056.
Frontal sinus empyemas, surgical principles essential to cure, Barnhill, 736.
Frontal sinus operation without deformity, Beck, 1048.

GARGLES in treatment of ozena, Schmidt 849.
Gastroscopy, Jackson, 263.
Genito-urinary system and upper air passages, Sendziak, 852.
Glottic spasm in adult, nasal origin, Conlon, 258.

HEADACHES, nasal, result of hyperemia central nervous organ, Rethi, 849.
Hemorrhage, nasal, in leukemia, Mayer, 1050.
Hydrocephalus, acute internal, secondary to streptococcal infection of labyrinth, Scott, 846.
Hyperinflation with Eustachian catarrh Braislin, 1030.

INTERNAL ear, double-sided necrosis with fatal hemorrhage, Benjamin, 1039.
Internal ear, non-suppurative diseases, vertigo and disturbances, Wittmaack, 242.
Intracranial complication of acute sphenoidal sinus suppuration, Trautman, 1051.
Intracranial complications of chronic recurrent suppurative otitis media Smith.
Intracranial complications of diseases accessory cavities nose, Coffin, 258.
Intracranial complication middle ear suppuration, Kopetzky, 117.
Intracranial complications otitic disease, ocular symptoms, Fridenberg, 140.
Intracranial disease otitic origin, Kanasugi, 1044.
Intratympanic operations under local anesthesia, Ballin, 249.

JUGULAR and sigmoid sinus thrombosis, double mastoiditis, Oppenheimer, 1026.
Jugular bulb phlebitis complicating mastoiditis, McKernon, 1027.
Jugular bulb, surgical exposure, Voss, 244.
Jugular vein, internal, excision, Dench, 1028.
Jugular vein, internal, phlebitis complicating mastoiditis, McKernon 1027.
Jugular vein, internal, thrombosis, operation, Allport, 333.

KELOID scalp and lobule ear, recurrent, Cocks, 1027.

LABYRINTH, acute suppuration following acute otitis media, Dench, 746.
Labyrinth functions, non-acoustic, Fridenberg, 670.
Labyrinth, histologic changes tympanic wall following radical mastoid operation Ruttin, 1042.
Labyrinth inflammations extending from middle ear, pathologic anatomy, Lange, 496.
Labyrinth, streptococcal infection, causing acute internal hydrocephalus, Scott, 846.
Labyrinth suppuration, clinical aspect, Neurnberg 1041.
Labyrinth suppuration, diagnosis and treatment, Canfield, 451.

Labyrinth suppuration, histology, Ruttin, 1040.
Labyrinth suppuration, prognosis of operative opening, Freytag, 246.
Labyrinthine deafness, chronic progressive Manasse, 244.
Labyrinthitis, acute, Alexander, 498.
Labyrinthitis due to meningitis, Yearsley, 249.
Labyrinthitis, infective, treatment Jansen, 367.
Lactic acid bacilli cultures in nose, throat and ear work, Curtis, 854.
Lamp for laryngoscopy, tracheoscopy and esophagoscopy, Horn, 853.
Laryngeal crises early symptoms of tabes, Freer, 1067.
Laryngeal growths removed by direct laryngoscopy, McCoy 1066.
Laryngeal nerves paralysis of peripheral origin, Delavan, 631.
Laryngeal pachydermia, Albrecht 1071.
Laryngeal syphilis, Heryng, 1068.
Laryngeal tuberculosis, Robinson, 850.
Laryngeal tuberculosis, significance for treatment tuberculosis pulmonum, Burger, 502.
Laryngocele, pathology, Scheren, 850.
Laryngoscopy, direct, laryngeal growths removed, McCoy 1066.
Laryngoscopy, lamp, Horn, 853.
Laryngospasm adults Neufeld, 259.
Larynx, giant tumors, Massei, 931.
Larynx papilloma in children, Clark, 787.
Larynx, primary lupus, Rosenberg, 1067.
Larynx, recurrent and abductor paraylsis—
Casselberry, 607.
Gleitsmann, 587.
Larynx tuberculosis, amputation epiglottis Moeller, 1064.
Lateral sinus thrombosis, Hammond, 1044.
Leptomeningitis purulenta following mastoiditis, Bryant, 163.
Leukemia, histologic changes in nasal mucosa and cause of nasal hemorrhages Mayer, 1050.
Lingua villosa nigra, Blegvad, 753.
Lupus larynx, primary, Rosenberg, 1067.
Lymphosarcoma tonsil, Goodale, 1058.

MASTOID complications of chronic recurrent suppurative otitis media, Smith, 733.
Mastoiditis and complications, McKernon, 1027.
Mastoiditis, complicated by typical and atypical cases sinus thrombosis Dabney 439.
Mastoiditis, double, with sigmoid sinus and jugular thrombosis, Oppenheimer, 1026.
Mastoiditis, followed by thrombosis posterior cerebral veins and artery, encephalitis, purulent leptomeningitis, lateral sinus phlebitis and parietal thrombosis, Bryant, 163.
Mastoiditis in the aged, Alderton, 1029.
Mastoiditis, two interesting cases, Crawford, 1036.
Mastoiditis with brain complications, Krause, 843.
Mastoid operation, radical, Allport, 250.
Mastoid operation, radical, for cure of chronic purulent otitis media, present status, Phillips, 1031.
Mastoid operation, radical, retroauricular opening, Winckler, 498.
Maxillary empyema caused by foreign bodies in nose, Krebs, 1056.
Maxillary sinusitis and osteitis, Moure and Brindel, 1053.
Maxillary sinus, local anesthesia in operations, King, 178.
Membrana tympani non-perforated pulsations, Fiedler, 1058.
Meniere's disease, myth and mystery, Gould, 1036.
Meningitis causing labyrinthitis, Yearsley, 249.
Meningitis of otitic origin, Kerrison, 847.
Meningitis purulenta complicating mostoiditis McKernon, 1027.
Middle ear inflammations extending to labyrinth, pathologic anatomy, Lange 496.
Middle ear cholesteatoma, Zimmerman, 848.
Middle ear discharge, treatment and prognosis Ruppert, 849.
Middle ear, granulation tissue removal followed by sinus thrombosis, Dench, 1048.
Middle ear sarcoma, Zebrowski, 847.
Middle ear suppurations, Bier's hyperemia, Isemer, 499.
Middle ear suppuration, intracranial complication, Kopetzky, 117.
Middle ear suppurations treated without removal drum membrane and ossicles, Heath, 250.
Morgagni ventricle acute edematous eversion, Reardon, 259.
Mouth, pigmentations of mucous membrane, Anthony, 1062.

NASAL analgesia as prognostic symptom dry catarrhal deafness Roy, 1033.
Nasal cavities, foreign bodies, Krebs 1056.
Nasal diphtheria primary—
Hubbard, 171.
Stimson, 256.
Nasal dysmenorrhea, Kuttner, 850.
Nasal headache result of hyperemia central nervous organs, Rethi, 849.
Nasal hemorrhage in leukemia, cause, Mayer, 1050.

Nasal mucosa, tuberculosis, Caboche, 180.
Nasal origin of glottic spasm in adult, Conlon, 258.
Nasal reflexes, Koblanck, 849.
Nasal septum, submucous resection, Killian, 502.
Nasal sporozoon, Wright, 257.
Nasopharyngeal polyp, hairy, congenital origin, Levinger, 851.
Neuroses nose, Grayson, 1056.
Nose, accessory cavities and optic nerve, Onodi, 1.
Nose and stomach diseases, connection, Hecht, 849.
Nose, conservatism in treatment of affections, Myers 167.
Nose diseases in children, questionable influence diathetic conditions, Kerley, 257.
Nose, malignant tumors, operative treatment, Denker, 959.
Nose, primary tumors sinuses, Citelli, 1055.
Nose work, lactic acid bacilli cultures, Curtis, 854.

OCULAR symptoms of intracranial complications otitic disease, Fridenberg, 140.
Optic nerve and accessory cavities nose Onodi, 1.
Oral cavity as factor in development of speech, Hudson-Makuen, 263.
Orbital complications of acute sphenoidal sinus suppuration, Trautman 1051.
Orbital complication of diseases nasal accessory sinuses, surgical treatment, Knapp, 1045.
Osteitis and maxillary sinusitis, Moure and Brindel, 1053.
Osteoma in faucial tonsils, Grosvenor, 470.
Otalgia, Hunt, 247.
Otitic brain abscess, diagnosis, Heimann, 800.
Otitic brain abscess operation, Stucky, 427.
Otitic disease ocular symptoms of intracranial complications, Fridenberg, 140.
Otitic origin of intracranial disease, Kanasugi, 1044.
Otitic origin of meningitis, Kerrison, 847.
Otitic sinus thrombosis and pyemia, Alexander, 1030.
Otitic thrombosis sigmoid sinus and internal jugular vein operation, Allport, 333.
Otitis interna acuta Stillman, 1036.
Otitis, chronic purulent, followed by sinus thrombosis, Knapp, 246.
Otitis media acuta followed by acute labyrinth suppuration, Dench, 746.
Otitis media purulenta chronica, Wiener, 1033.
Otitis media purulenta chronica, associated with brain abscess, Page, 1034.
Otitis media purulenta chronica, present status radical mastoid operation for cure, Phillips, 1031.
Otitis media purulenta chronica, recurrent, and complications, Smith, 723.
Otosclerosis, etiology, Harris, 645.
Otosclerosis, pathology and prognosis, Bryant, 652.
Otosclerosis, symptoms and diagnosis, Shambaugh, 661.
Ozena, relation to syphilis, Frese, 1051.
Ozena, submucous resection, Mermod, 995.
Ozena, treatment by gargles, Schmidt, 849.

PACHYDERMIA laryngis, diffuse, Albrecht, 1071.
Pansinusitis nasalis with fatal course, Struycken, 499.
Papilloma larynx in children, Clark, 787.
Pathology suppurations temporal bone, Wales, 398.
Pharyngeal syphilis, Heryng 1068.
Phonation center in cortex dog, Katzenstein, 1065.
Piano-string theory of the basilar membrane, Bryant, 847.
Pigmentations mucous membrane mouth, Anthony, 1062.
Pleuritis, importance upper respiratory tract in the etiology of cryptogenetic infections, Wood, 855.
Polyp, bleeding, septum, Trautman, 1057.
Pulsations in non-perforated membrana tympani, Fiedler, 1038.
Polyp, hairy nasopharyngeal, congenital origin, Levinger, 851.
Pyemia and otitic sinus thrombosis, Alexander, 1030.

RECURRENT laryngeal paralysis Casselberry, 607.
Recurrent laryngeal paralysis, anatomy and physiology, Gleitsmann, 587.
Recurrent laryngeal nerves, paralysis peripheral origin, Delavan, 631.
Respiratory tract, upper, relation to cryptogenetic infections in relation to pleuritis, Wood, 855.
Respiratory tract, upper, tuberculosis, Wood, 261.
Rhinology, ligation external carotid, Jackson, 261.
Roentgen rays in rhinology, Scheier, 1052.

SARCOMA middle ear, Zebrowski, 847.
Scalp, recurrent keloid, Cocks, 1027.
Septal operation, submucous, Freer, 1057.
Septum, bleeding polyp, Trautman, 1057.
Sigmoid sinus and jugular thrombosis, double mastoiditis, Oppenheimer, 1026.
Sigmoid sinus phlebitis complicating mastoiditis, McKernon, 1027.
Sigmoid sinus thrombosis operation, Allport, 333.
Sinus phlebitis, following mastoiditis, Bryant, 163.

Sinus thrombosis after chronic purulent otitis complicated with cholesteatoma, Knapp, 146.
Sinus thrombosis complicated by cerebral abscess in temporosphenoidal lobes, Dabney, 844.
Sinus thrombosis complicating mastoiditis, atypical and typical, Dabney, 459.
Sinus thrombosis following removal granulation tissue from middle ear, Dench, 1028.
Sinus thrombosis, otitis, and pyemia, Alexander, 1030.
Sinuses nose, primary tumors, Citelli, 1055.
Sinusitis associated with serious eye complications, Wiener, 1049.
Sodium perborate—
  Heydenreich, 852.
  Hartman, 853.
Speech disturbance in extradural abscess, Jansen, 1050.
Speech, oral cavity as factor in development, Hudson-Makuen, 263.
Sphenoidal sinus empyema, radical operation and new instruments, Skillern, 1045.
Sphenoidal sinus suppuration with intracranial and orbital complications, Trautman, 1051.
Sporozoon, nasal, Wright, 257.
Stomach and nose diseases, connection, Hecht, 849.
Streptococcic infection pharyngeal adenoid tissue adult, Bryant, 1060.
Submucous resection nasal septum, Freer, 1057; Killian, 502.
Submucous resection in ozena, Mermod, 995.
Syphilis pharyngeal and laryngeal, Heryng, 1068.
Syphilis, relation to ozena, Frese, 1051.

TABES, laryngeal crises and paresis of abductors vocal cords, Freer, 1067.
Temporal lobe abscess causing anosmia, Bloch and Hechinger, 1054.
Temporal bone, causation suppuration, Sauer, 431.
Temporal bone, important surgical relations, Shambaugh, 250.
Temporal bone, pathology and bacteriology suppurative diseases, Wales, 398.
Temporosphenoidal lobe abscess, sinus thrombosis complicating, Dabney, 844.
Throat disease in children, questionable influence of diathetic condition, Keriev, 257.
Throat, local treatment acute inflammations, from standpoint pathology, Goodale, 851.
Throat work, bottle and bacilli cultures in, Curtis, 854.
Thrombosis lateral sinus, Hammond, 1044.
Thrombosis posterior cerebral veins and artery, etc., following mastoiditis, Bryant, 1044.
Thrombosis sigmoid sinus and jugular, double mastoiditis, Oppenheimer, 1026.
Thrombus, do not remove, Voss, 1029.
Tone impressions, sensitiveness cochlea, Bryant, 1044.
Tone perception, problem, Shambaugh, 251.
Tongue, black hairy, Blegvad, 753.
Tongue, black, hairy, pathologic anatomy and etiology, Haenisch, 1061.
Tongue, deep-lying abscesses, Frank, 854.
Tonsil, applied anatomy, Barnes, 1057.
Tonsil, chondroma and osteoma in, Grosvenor, 470.
Tonsil, lymphosarcoma, Goodale, 1058.
Tonsil operations, results, Clark, 1058.
Tonsillolith, case, Anders, 1060.
Tonsillotomy, new method of relief, hemorrhage, Henkes, 505.
Torticollis complication adenotomy, Neufeld, 1061.
Tracheoscopy, diagnostic and therapeutic relation, Schmiegelow, 1064.
Tracheoscopy, lamp for, Horn, 853.
Tuberculosis, ear, Bryant, 1035.
Tuberculosis, larynx, Robinson, 850.
Tuberculosis larynx amputation epiglottis, Moeller, 1061.
Tuberculosis larynx, significance for treatment tuberculosis pulmonum, Sanger, 502.
Tuberculosis nasal mucosa, Caboche, 180.
Tuberculosis pulmonum, significance tuberculosis laryngis for treatment, Sanger, 502.
Tuberculosis upper air passages, treatment, Heryng, 1060.
Tuberculosis upper respiratory tract, Wood, 261.
Tumors sinuses nose, Citelli, 1055.
Turbinals, morphology, Ingersoll, 991.
Turbinate, indications resection of middle, Casselberry, 258.
Turbinate, purulent bone cyst inferior, Cohn, 502.

VERTIGO in non-suppurative diseases internal ear, Wittmaack, 243.
Vocal cord abductors paralysis early symptom tabes, Freer, 1067.

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